The Journal of Thoracic Surgery

VOL. 8 APRIL, 1939 NO. 4

Original Communications

LOBECTOMY AND PNEUMONECTOMY IN PULMONARY TUBERCULOSIS*

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CERTAIN cases of phthisis are cured only by a partial or total surgical removal of the diseased lung. At the present time we believe that pulmonary lobectomy and pneumonectomy should be included in our armamentarium in the modern program of surgical collapse therapy. It still remains definitely a debatable subject; the indications and choice of suitable cases are yet to be worked out.

The historical aspect of lobectomy and pneumonectomy in tuberculosis is not a drab tale. On the contrary, it was an heroic and dramatic series of events. In 1881 Block,[1] of Danzig, who two years previously reported his work on experimental pneumonectomy, resected the apices of both lungs in one operation, and then, upon the death of his patient (a female relative at whose insistence he had operated), was prompted to suicide without recording detailed observations of his patient. Walton[2] reports in the *Boston Medical and Surgical Journal* that the medicolegal inquest disclosed that the patient did *not* have tuberculosis.

Ruggi,[3] in 1885, operated upon two patients with tuberculosis that were "in a deplorable state." The first patient, 30 years old, had positive sputum of five years' duration, fungus synovitis of the knee, intestinal tuberculosis, and a fist-sized apical cavity on the right. The contralateral lung was apparently clear. As the second and third ribs were being resected, a tear was made into the cavity. Partial removal of the cavity wall was accomplished and the remainder was curetted. A dozen forceps were left in the wound at the completion of the two-hour operation. The patient succumbed on the ninth day.

*Read before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

Ruggi's second case, 32 years old, had tuberculosis for one year, repeated hemoptyses and a right apical lesion. "The parietal pleura could not be freed from the lung; therefore the procedure, commenced in such good faith, was abandoned. Two days later the patient died. It is true that his situation before operation was desperate and that postoperatively he refused all care."

Tuffier[4] was not discouraged by this precedent since, in his opinion, the extent of disease precluded cure. As a prosector, he had studied carefully the anatomical aspects of removing the apex of the lung. He purposed removing tuberculosis at its debut: a single and unilateral nodule without evidence of other involvement. In 1891 Tuffier[4] resected through an anterior intercostal incision the apex of the lung 6 cm. below its upper surface. The resected apex contained a tuberculous nodule the size of a small nut which was surrounded by disseminated tubercles, but there was no cavity. The patient, a 22-year-old male, had an uneventful course and was presented a month later to the Surgical Society, and was presented as cured, in 1895, to the Congress of Surgery.

In 1893 Lowson,[5] using a double chained ligature, resected a fist-sized apex of the right lung in a 34-year-old patient. A drained circumscribed hemopyothorax somewhat retarded the eventual cure. Doyen[6] similarly reported in 1895 the extirpation of a portion of a lobe in a child of ten years. His patient at that time had been cured more than two years and presented no other tuberculous lesion.

Contrary to the reports of Nissen,[7] Garré and Quincke,[8] Macewen,[9] in 1895, did not attempt pneumonectomy but rather drained a huge tuberculous cavity at the first operation, and in a subsequent stage resected shreds of pulmonary tissue. The adherent apex of the lung was left intact. This case scarcely should be considered a true lobectomy.

Interest apparently waned in this particular phase of tuberculosis surgery for there is no case reported during the next thirty years. Quoting Tuffier,[4] "These different attempts have not been approved by our colleagues, if I except Roswell Park.[10] One of them has objected because of the primary diffusion of the lesions, the anatomical pathology not yet established, and the medical cure is frequent and the impossibility of exact diagnosis. I do not despair of seeing my colleagues recall their ostracism and accept pneumonectomy as good for certain forms of pulmonary tuberculosis, especially the truly localized early tuberculosis."

While we do not prescribe the above statement, nor can we agree entirely with many of Tuffier's teachings, we are forced to admire his surgical enthusiasm, and admit that his surgery and writings have been inspiring contributions in the advance of thoracic surgery. We must not lose sight of the fact that much of the pathological physiology of pulmonary disease which is known to us, as well as the laboratory and roentgen facilities, was not available some fifty years ago when this pioneer work began.

With interest and enthusiasm for the use of lobectomy and pneumonectomy in bronchiectatic and tumor cases steadily increasing during the last fifteen years, a revival of the procedure for tuberculosis eventually was inevitable. Freedlander,[11] in 1934, successfully resected the right upper lobe for a tuberculous cavity which failed to close following an apparently perfect pneumothorax. After several months the patient was discharged from the sanatorium, an apparently arrested case.

In 1935 Eloesser[12] and O'Brien[13] each reported a case of one-stage lobectomy for tuberculous cavities. Tuberculous spread caused death in both instances. Beye,[14] in 1934, inadvertently but successfully resected the right lower lobe of the lung for saccular bronchiectasis which on microscopic study proved to be tuberculosis. In 1935 Beye[14] successfully removed a tuberculous excavated lung which failed to heal following multiple thoracoplasties. Convalescence in this case was excellent but at last report the patient had a patent bronchocutaneous fistula.

In 1936 Lindskog[15] under an erroneous diagnosis unwittingly performed a left total pneumonectomy on a tuberculous bronchiectatic lung. A year later the patient had tuberculous infiltration of the opposite lung, was febrile, and the fistula was still patent with positive bacillary drainage.

These few cases are scarcely adequate from which to draw conclusions regarding indications and contraindications in lobectomy and pneumonectomy for tuberculosis. Increasing experience will, no doubt, eliminate many cases which we now consider fair prospects for radical surgery. Our own opinion regarding indications we will withhold until after the presentation of our series of four cases.

CASE 1.—M. B., 32 years old, Mexican gardener, had been ill for six years. Illness began in 1929; weakness, weight loss, chest pain, and regularly recurring hemoptyses. His wife and three children were alive and well. According to x-ray (Fig. 1*A*) and physical findings he had a persistent 2 cm. right apical tuberculous cavity. Extended sanatorium treatment along with pneumothorax (Fig. 1*B*), closed pneumonolysis, phrenicectomy, and scaleneotomy (Fig. 1*C*) failed to arrest the ever-increasing hemorrhages which severely depleted the patient.

It was felt that thoracoplasty would yield no better collapse than the already existing pneumothorax. Furthermore, since his hemorrhages had increased to 600 c.c., and once 800 c.c. at a time, we feared fatal hemorrhage on the operating table if thoracoplasty were done; whereas, in lobectomy it is a matter of only a few minutes before the hilar stump ligation controls all bleeding. When he came to surgery his hemoglobin was down to 40 per cent in spite of several small transfusions. He had positive sputum and bronchoscopy showed that the blood was coming from the right upper lobe. On July 22, 1935, a right upper and middle lobectomy was done through an intercostal posterolateral approach and the chest was closed without drainage. Convalescence was uneventful and he was discharged from the Cedars of Lebanon Hospital to the Sanatorium on the fourteenth postoperative day (Fig. 1*D*).

Four months later at the Olive View Sanatorium a two-stage eight-rib extrapleural thoracoplasty was completed to obliterate the pneumothorax space, which did not become infected although the bronchus opened into it.

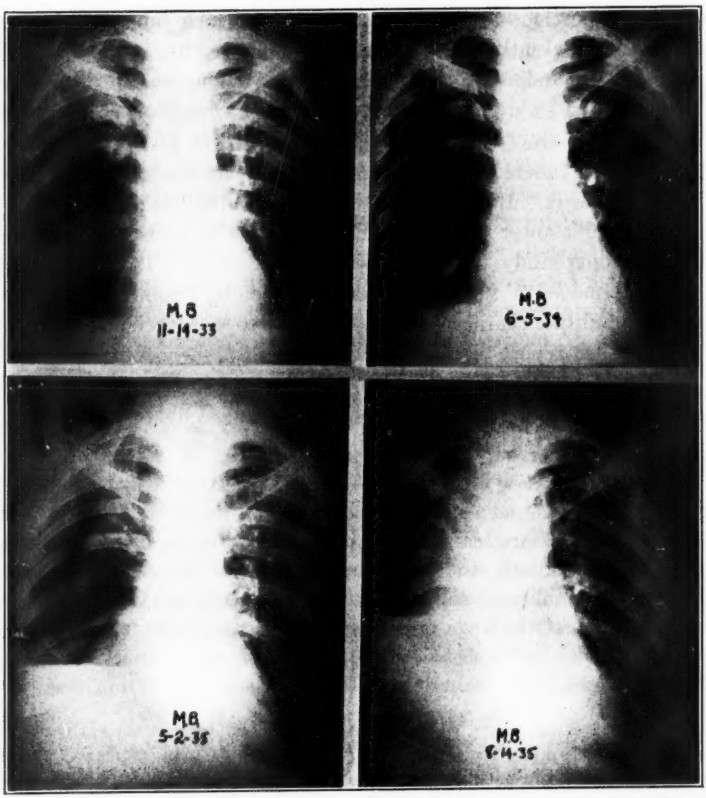

Fig. 1.—Case 1. *A*, Right apical cavity behind first costochondral junction; *B*, after six and one-half months' right pneumothorax and three months' temporary diaphragmatic paralysis without relief; *C*, after thoracoscopy and right scaleniotomy and permanent phrenicectomy; *D*, twenty-three days after double (upper-middle) lobectomy.

The specimen (Fig. 2) shows an apical cavity filled with laminated old and new blood clot, and opening into it a large branch of the pulmonary artery. Microscopic sections taken at one edge of the cavity show new blood clot about the old organized laminated blood clot which every four days, as it retracted from the mouth of the leaking vessel, caused regularly recurring hemoptyses. Scattered tubercles were seen throughout the lung.

The patient has been entirely well for over two years, without cough or sputum (Figs. 3 and 4). The wounds remained clean throughout all his surgery. He works and supports his family after seven years in sanatoria.

Fig. 2.—Case 1. Specimen of right upper and middle lobe. Apical cavity filled with new and old laminated blood clot.

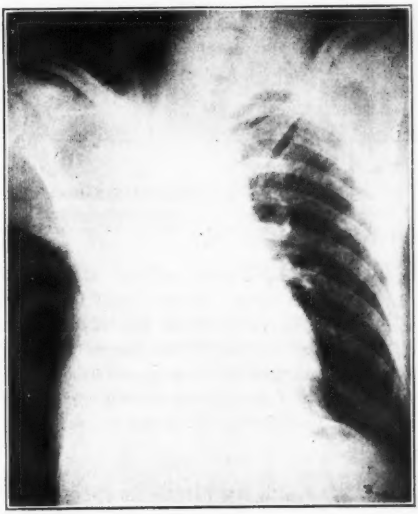

Fig. 3.—Case 1. Roentgenogram one year following eight-rib extrapleural thoracoplasty.

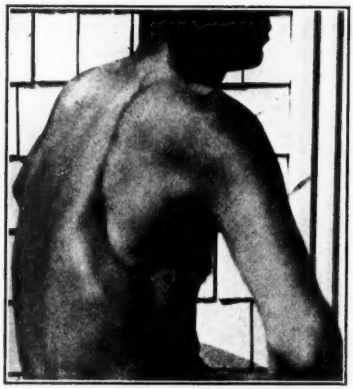

Fig. 4.—Case 1. The patient two years following lobectomy.

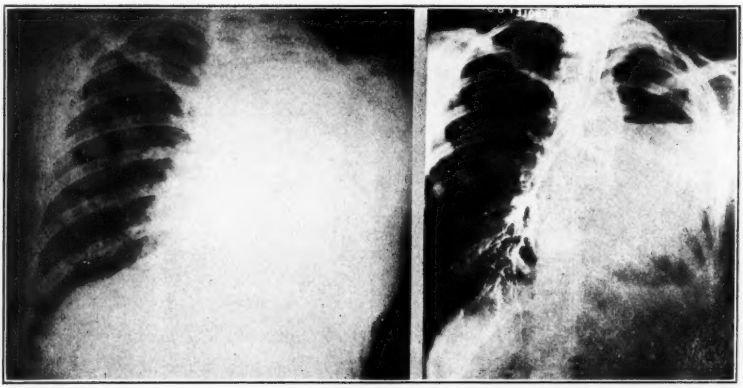

Fig. 5. Fig. 6.

Fig. 5.—Case 2. Roentgenogram on admission to sanatorium, 1933.
Fig. 6.—Case 2. Bronchogram (Dec. 24, 1935) demonstrating marked stenosis of left main stem bronchus proximal to upper lobe bronchus and pleural effusion.

CASE 2.—L. W., 28 years old, housewife, had been ill for over four years. Onset of symptoms came in 1932, with cough, fever, weight loss, and expectoration positive for tubercle bacilli. She was admitted to the Olive View Sanatorium in 1933 after several months' bed rest elsewhere. The patient had a persistent harassing cough, daily fever of 102 and upward, expectoration of twelve ounces daily of purulent sputum, weight loss of forty pounds during two and one-half years in the Sanatorium. X-ray examination (Fig. 5) revealed total atelectasis of left lung with retraction to the left.

Pneumothorax, phrenicectomy, and closed pneumonolysis failed to relieve symptoms and she developed an effusion. Bronchography (Fig. 6) revealed stenosis of the left main stem bronchus, confirmed by bronchoscopy to be a stenosing tuberculous tracheobronchial lesion just beyond the carina. The lung distal to the stenosis was being destroyed by suppuration, presumably caused by poor drainage, and since collapse was of no benefit the pneumothorax was abandoned.

We proposed extirpation of the lung as the procedure of choice since thoracoplasty offered little toward eventual cure. Therefore, on June 18, 1936, through an intercostal posterolateral approach a tourniquet pneumonectomy was done at the Los Angeles County Hospital. One tuberculous caseous subpleural abscess the size of a hen's egg was encountered in the first rib circle. The wound was closed with intercostal airtight drainage posteriorly. Later it was necessary to convert the drainage into an open one by rib resection and the insertion of a larger drain tube.

This patient likewise had an unusually smooth postoperative course with lower temperature immediately after surgery than in two and one-half years in the sanatorium. The wound healed per primum but there persisted a bronchial fistula through the site of drainage. She gained forty pounds in weight and was discharged home after three months' hospitalization. The right lung had remained clear throughout.

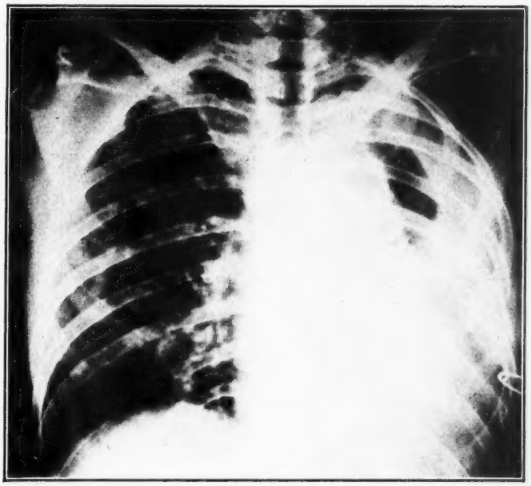

Fig. 7.—Case 2. Acute exudative spread occurred five months after pneumonectomy.

Approximately eight weeks after discharge, she returned to the hospital with an acute exudative lesion (Fig. 7) in the remaining lung, which has necessitated her return to the sanatorium. This lesion, however, has not excavated and in the past year has shown progressive clearing. A small residual empyema with persistent fistula remains.

Specimen (Fig. 8) was that of a contracted indurated lung with thickened dilated bronchi and multiple abscesses. Scattered throughout the lung were tubercles.

The patient (Fig. 9) has no cough and raises practically nothing and, in spite of her activation due to her activity on the outside, she shows progressive improvement and has maintained her forty-pound gain in weight.

Case 3.—E. A., 29 years old, schoolteacher, came to the Barlow Sanatorium, Dec. 1, 1935, with symptoms of tuberculosis for five months. At that time she had a large right apical cavity with diffuse infiltration throughout the upper lobe (Fig. 10*A*). With bed rest and inadequate pneumothorax the disease progressed (Fig. 10*B*), and, although somewhat improved later by a temporary phrenicectomy, an extensive thoracoplasty was advised and completed in September, 1936.

Fig. 8.—Case 2. Cut surfaces of upper and lower lobes of lung revealed multiple caseous abscesses with thickened dilated bronchi.

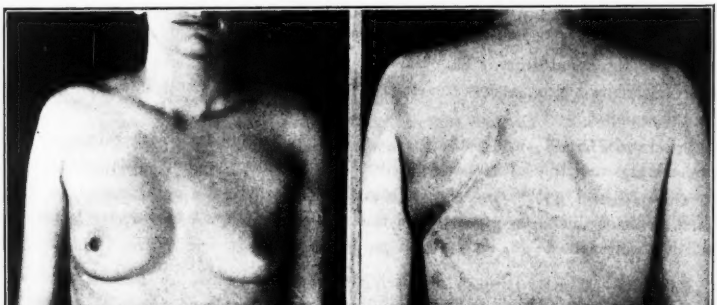

Fig. 9.—Case 2. Postoperative photograph of patient.

The patient, although vastly improved, still had a residual cavity (Fig. 10*C* and *D*) with a daily four-dram positive sputum output. The stiff-walled cavity had receded in against the mediastinum mesial to the spine and had not changed for months. The diaphragm had returned to function. Bronchoscopy did not reveal tuberculous tracheobronchitis. Since she had a good collapse by thoracoplasty, it

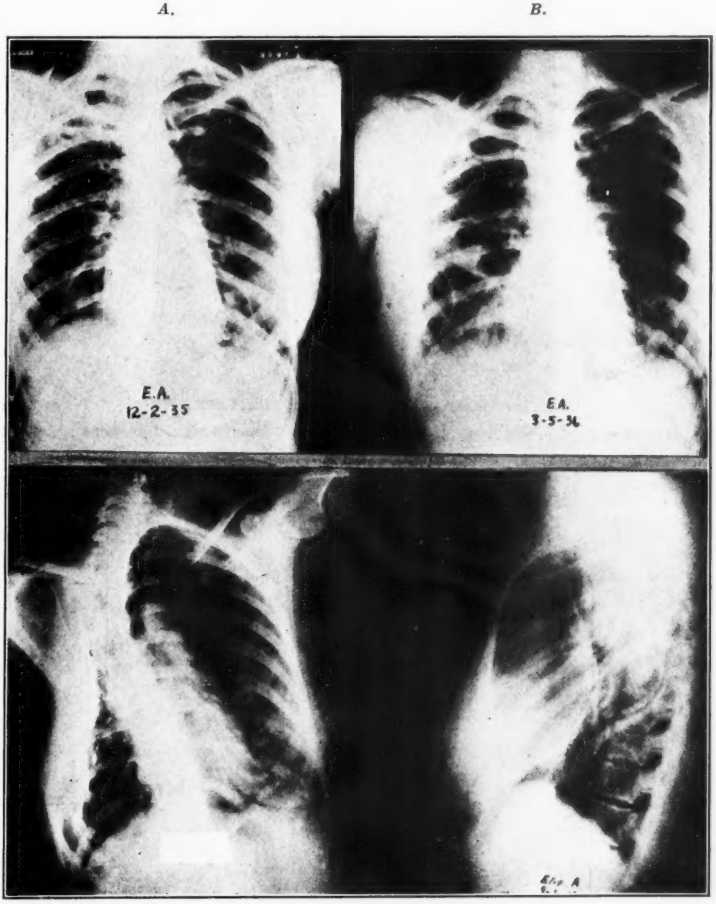

Fig. 10.—Case 3. *A*, Roentgenogram on admission to sanatorium; *B*, after three months' bed rest and inadequate pneumothorax; *C*, slightly oblique roentgenogram showing residual cavity underlying mesial transverse process and body of fifth dorsal vertebra; *D*, lateral roentgenogram showed cavity just anterior to spine.

was felt that upper lobectomy was indicated to eradicate the cavity, it being unlikely that revisional thoracoplasty would accomplish cavity closure and persistently negative sputum.

Right upper lobectomy was carried out Aug. 9, 1937, at the Hospital of the Good Samaritan. The patient actually had less reaction to operation than any one

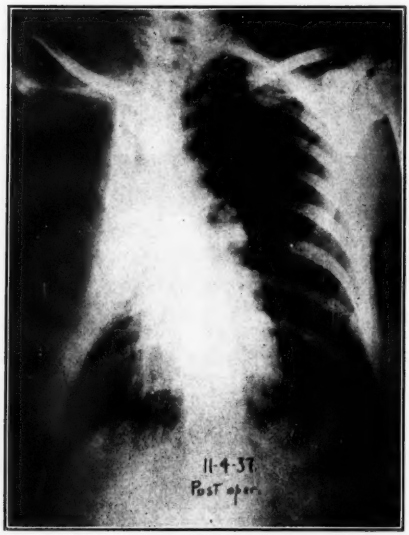

Fig. 11.—Case 3. Roentgenogram four months after lobectomy. Note good aeration of right lower lung.

Fig. 12.—Case 3. Specimen right upper lobe with superficial stiff-walled tuberculous cavity.

of the stages of her thoracoplasty, and the convalescence was uneventful. She is without cough and sputum, looks and feels well, has gained thirty pounds since operation on continued sanatorium care, and the wound has healed except for a bronchocutaneous fistulous tract which is progressively becoming smaller with little drainage. All postoperative roentgenograms show no active tuberculosis (Fig. 11). The patient was discharged home from the sanatorium April 2, 1938.

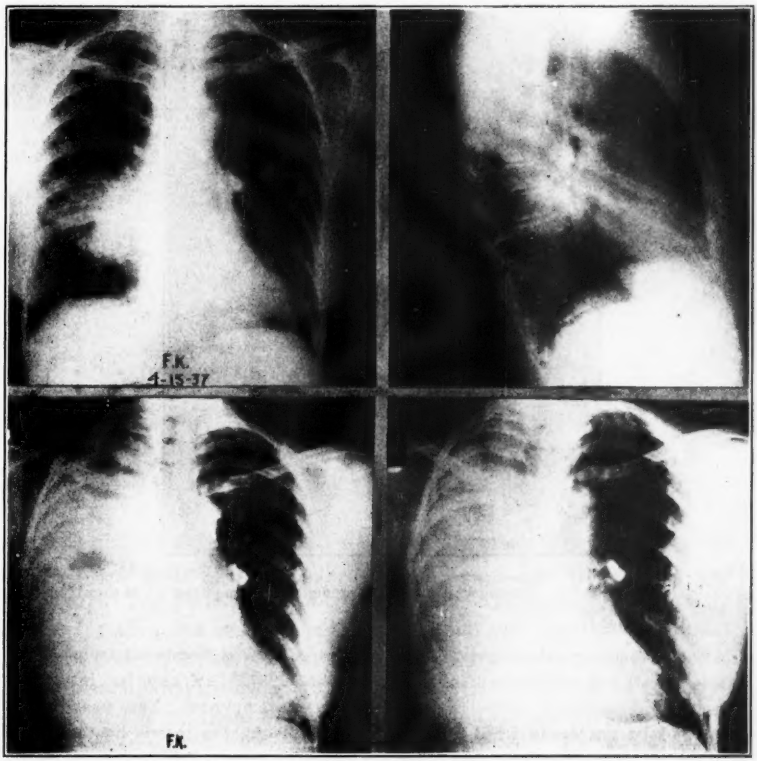

Fig. 13.—Case 4. *A* and *B*, preoperative roentgenograms showing mass and infiltration in middle third of right lower lobe; *C*, roentgenogram five days postoperative showing a high right diaphragm with multiple fluid levels above it; *D*, roentgenogram four months postoperative shows complete opacity and retraction of trachea.

The specimen (Fig. 12) shows the cavity close to the surface. During the freeing of the lung from the chest wall a tear was made into the cavity. The cut surface shows the thick-walled persistent tuberculous cavity.

CASE 4.—F. K., 49 years old, housewife, in February, 1937, had so-called "influenza" from which she did not seem to regain her previous good health. At the time of acute illness she had nonproductive cough, fever, malaise, and discomfort in the right chest; there was no hemoptysis. Fever subsided on few days' rest but cough persisted with fifteen-pound weight loss in two and one-half months. There

was occasional clear mucoid sputum, negative on smears for tubercle bacilli on three occasions. X-ray examination (Fig. 13) revealed mass in the right lower lobe. Her physician referred her to a bronchoscopist who did not find positive evidence of malignancy but believed the narrowing and fixation of the lower lobe branch bronchi very suggestive of malignancy and advised deep x-ray therapy. She was then referred for this treatment but the roentgen therapist felt that an exploration with a view of pneumonectomy was indicated before therapy was advisable. She was, in turn, referred to us for surgery.

Fig. 14.—Case 4. Specimen of entire right lung. Right lower lobe sectioned toward its hilar aspect. There was no gross evidence of caseation on the cut surface of the "tuberculoma."

After preliminary pneumothorax for five days, a total right pneumonectomy was completed at the California Hospital, June 26, 1937. An anterior intercostotranschondral approach, described by Overholt, was employed. The mass in the posterior lobe was easily freed from the parietal pleura, the vessels ligated singly with silk, the lung amputated just below the carina, the main stem bronchus crushed and doubly sutured with an atraumatic needle and chromic catgut, and then the stump buried in the mediastinum. There was closure without drainage; six anthracotic glands were encountered and removed.

Her postoperative course was uneventful. She was aspirated five days postoperatively and 200 c.c. of blood-tinged sterile fluid obtained. The wound healed per primum and she was discharged home on her seventeenth postoperative day, still having afternoon fever of 99.5 which persisted for about a month after return home, but she needed no further aspirations. Her x-ray photographs (Fig. 13*C* and *D*) are those of a typical uncomplicated postoperative pneumonectomy.

The specimen (Fig. 14) grossly was that of a tumor mass the size of a tennis ball in the posterior lobe. No smears were taken at the time but microscopic examination revealed we had removed a tuberculous lung; the report came back "tuberculoma," somewhat to our chagrin.

However, the patient has had a most fortunate result. She has had no fever, cough, or sputum, and after a period of four months' bed rest has been allowed to increase her privileges to be out and about. She has regained much of her previous strength. The chest is somewhat retracted on the right, but she had neither fistula nor infection (Fig. 15).

This case, of course, is presented as a matter of interest and not as an indication for pneumonectomy.

Fig. 15.—Case 4. Photograph six months postoperative. Slight retraction of chest but surprisingly little deformity.

It is difficult to assume, and we believe it impossible to accomplish, a complete ablation of tuberculosis of lungs by lobectomy or pneumonectomy. The pathology involved and the very nature of the disease in these far-advanced cases of phthisis preclude such surgical attainments. We do believe, however, that there is a place for such operations and have presented three cases with their indications for radical surgery.

This brings us, then, to the question of indications for lobectomy and pneumonectomy in tuberculosis of lungs. Where the partial or complete removal of a tuberculous lung offers the patient his only chance of cure after other recognized and indicated procedures have failed to bring about the desired result, and if the patient's general condition apparently is suitable for major surgery, with presumably good resistance to his disease, this may be considered a fair indication.

In patients having frequent huge hemorrhages which were not controlled by the collapse of a pneumothorax and a complementary phrenicectomy, I am, boldly perhaps, of the opinion that lobectomy is a far safer procedure than thoracoplasty. In the interim between stages of thoracoplasty in these severe bleeders fatal pneumonic contralateral spread of disease too often occurs. It is ridiculous to seek solace in the time-worn assumption that without thoracoplasty the patient would

have had the same eventual outcome, namely, a spread or drowning in his own blood. By lobectomy the source of the bleeding is controlled in fifteen minutes; on the other hand, it may not be appreciably influenced by thoracoplasty in these firm indurated lobes.

Let us consider suppuration of lung complicating tuberculosis whether the drainage of the affected lung be good or poor. A suppurating lung is not cured by thoracoplasty; in fact, many times it is made worse by virtue of disturbance of bronchial drainage. Toxicity is usually increased by the burden of any surgery except that removing the source of the toxicity. Why then try to collapse a boggy, hard, indurated shell of a lung that is virtually a cesspool? Lobectomy or pneumonectomy is certainly the procedure of choice.

There is also the problem of the tuberculous cavities that failed to close with extensive thoracoplasty, in some cases their stiff walls receding mesial to the spine. We have come to believe that regardless of how many thoracoplasties be repeatedly revised, a certain number of cavities will persist with positive sputum. If these cases were reasonable risks for thoracoplasty, lobectomy for residual cavity should be given due consideration. If he chooses to accept the risk of surgery, the patient should not be deprived of his chance to return to society arrested of his tuberculosis.

Another type of lesion that might eventually come to lobectomy is one having an atelectatic firmly contracted honeycombed lobe, with persistent positive sputum after thoracoplasty has been completed. Many of these indurated lobes are not collapsed by extensive thoracoplasty but merely are changed in position, being dropped downward and mesially against the mediastinum. Months or years of bed rest usually do not influence the course and revisional thoracoplasty often fails to render persistently negative sputum.

There is also the rare unilobar basilar tuberculous cavity which does not heal after the usual procedures of collapse therapy have been employed. Where pneumothorax and/or phrenicectomy, with or without pneumoperitoneum, have failed, thoracoplasty does not offer the assurance of cavity closure as it does in lesions of the upper lung. Lobectomy should be considered in such cases if the patients are good or fair risks for major surgery. If by pneumothorax the upper lobe is free, it would be well to encourage its adherence to the parietal pleura either by pleural poudrage (Bethune) or by irritation with dry gauze at the first stage of lobectomy. Should the general condition of the patient preclude lobectomy, cavity drainage would be considered.

No doubt as time goes on and our experiences increase, there will be many more indications for this type of surgery. For years we have been laboring under the false premise that a tuberculous lung should not be touched. While this is true in the great majority of cases, we must bear in mind the occasional exceptions to the rule and be not too dogmatic in our beliefs.

SUMMARY

1. Four cases of lobectomy and pneumonectomy for tuberculosis without mortality have been presented; two of the patients are well and two are greatly improved.

2. Indications for radical surgery have been briefly discussed.

3. Obviously no definite conclusions can be drawn from a small number of cases; we believe, however, that there is a place for lobectomy and pneumonectomy in our surgical treatment of pulmonary tuberculosis.

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DISCUSSION

DR. EVARTS A. GRAHAM, ST. LOUIS, MO.—Of course all of you know that the first resections of portions of lung tissue were done for tuberculosis when Lawson, an Englishman, about 1881, removed the upper lobe of the lung successfully for tuberculosis, and Tuffier, about 1890, removed the upper lobe of the lung or at least the apex of the lung for tuberculosis. There were perhaps a few other scattered successful operations of this type, but Paget emphasizes in his book on Chest Surgery, written in 1896, the large number of tragedies which resulted from the attempted removal of pulmonary tissue for tuberculosis, including the suicide of one unnamed surgeon who lost two patients as a result of this operation, and states that the operation was abandoned.

Of course in those days really little was known about thoracic surgery, and even little was known about surgery in general, so we cannot draw conclusions from the early discouragements of that time and apply them to the year 1938. As a matter of fact, instead of throwing cold water on Dr. Jones's ideas as expressed here, I wish to add a bit of commendation. Fifteen years ago I removed a considerable portion of the right lung in a man at that time 60 years of age, on a mistaken diagnosis that it was suppurative disease and not tuberculosis; but I found to my astonishment on microscopic examination that it was tuberculosis, secondarily infected. This man was alive seven or eight years afterwards. I do not know what has happened to him in recent years. He was alive and nearly 70 years old the last time I heard from him.

In two other cases that Dr. Singer and I had together in which the diagnosis of suspected tuberculosis had been made but not positively proved, and in both of which repeated devastating hemorrhages had occurred, operations of lobectomy were carried out, with the thought that the patients were tuberculous subjects, in order to arrest what seemed to be fatal hemorrhage. Both of these patients recovered well from the operation but they were found not to have tuberculosis when we came to the microscopic examination of the lung.

Two years ago Dr. Singer and I reported before this Society three cases in which lobectomy had been done for calcified lesions in the lung, the nature of which we could not exactly diagnose. These were laminated calcified lesions which looked like tumors of the lung. Some of you will remember those cases. We have always felt, although there is no positive proof of it, that these cases really were cases of tuberculosis. Dr. Haight told me yesterday that he had recently had a similar case in which, if I understood him correctly, a positive diagnosis of tuberculosis was made.

Recently again, only a few months ago, under the mistaken idea that I was operating on a tumor, because we had no positive evidence of tuberculosis in this man, I removed the upper lobe for a lesion very similar to this tuberculoma which Dr. Jones described. He made an absolutely uneventful recovery, left the hospital in about three weeks for his home, about 1500 miles away, and has had no further trouble so far as the lobectomy is concerned. Again in another recent case I removed the left upper lobe because of intractable hemorrhage from bronchiectasis. Repeated sputum examinations before the operation were negative for tuberculosis but the examination of the removed specimen showed it to be tuberculosis. The patient made an uneventful recovery.

I would say, therefore, in the light of my own experience with what amounts to approximately five or six cases of lobectomy performed for either tuberculous or suspected tuberculous lesions, that the operation is one which probably has a place rarely. I do think it would be a mistake for us to go too fast in this. We are likely to be carried away by initial success. I hope, however, that no member of this Association will have to come to the point of committing suicide because of some tragedies in this sort of work.

DR. SIDNEY SHIPMAN, SAN FRANCISCO, CALIF.—I would like to present one case which we had at the University of California which came to us with a diagnosis of nontuberculous disease but was shown later to have tuberculosis.

This was a girl aged 28 years who came in with a diagnosis of nontuberculous disease; but it was noted, on lipiodol pictures she had had taken several months before because of the fact that she had cough and expectoration, that she had a stenosis of her stem bronchus on the right side just above the opening of the middle lobe bronchus. Her sputum had been examined repeatedly but had always been found negative. She was bronchoscoped and when the bronchoscope was introduced it was seen that the stem bronchus was almost occluded by a circular ring of scar tissue. When the sucker was introduced below this area, pus was removed, which was found later to be loaded with tubercle bacilli. Apparently secondary infection played very little part in this girl's illness. She was given pneumothorax and a temporary phrenic operation, but failed to improve.

Dr. Brunn then saw the patient and suggested she should have a lobectomy. Both the middle and lower lobes on the right side were removed, and the patient got along very well for the first few days after lobectomy. However, she developed a subpectoral abscess which had to be drained. Following that she did well and is now perfectly healthy. She never had a bronchial fistula. We think this is probably one of the indications for lobectomy in tuberculosis.

DR. ROBERT JANES, TORONTO, ONT.—I think perhaps it would be worth while confessing that we have done one lobectomy for tuberculosis, the indication

being the last Dr. Jones mentioned, a single cavity in the center of the lower lobe in a patient in whom pneumothorax was impossible and in whom phrenic paralysis failed to close the cavity.

Recovery was uneventful except for development of a very small empyema and a fistula which has subsequently closed. The patient is well and has certainly much less chest deformity than had she had a thoracoplasty, for that particular type of cavity is difficult to close.

So far as the first indication is concerned, that of hemorrhage, I would be inclined to question it. In the last two years I have performed thoracoplasty on three patients who had active, massive, recurring hemorrhage, and rather to my surprise they have all done very well, and the hemorrhage has been controlled after the first stage of thoracoplasty. It apparently does not necessarily require a lot of collapse to control the hemorrhage.

One has considered seriously on several occasions patients who presented the other two indications, and it seems to me that in the presence of residual cavities in patients who have had a thoracoplasty, or frequently thoracoplasties, where every effort has failed to effect a closure of the cavity, lobectomy might be considered; also, in those who have an infective process beyond a marked stenosis of the bronchus, it seems to me to be a reasonable suggestion.

DR. POL N. CORYLLOS,* NEW YORK CITY.—I want to congratulate Dr. Jones for his interesting communication and his brilliant results. We all agree that there is a place for lobectomy in the surgical treatment of pulmonary tuberculosis. Giant cavities persisting or even increasing after pneumothorax offer a great temptation for lobectomy. On the other hand in revision of thoracoplasties which have failed to collapse large cavities again lobectomy appears the reasonable procedure. Unfortunately there is the difficulty of the management of the tuberculous bronchus and of the danger of production of bronchial fistula leading to mixed infection tuberculous empyema.

I have performed lobectomy twice on tuberculous patients with unsatisfactory results.

The first case was a colored woman, 33 years old. She had a giant cavity of the right upper lobe which remained uncollapsed after three stages of thoracoplasty. During revision I was able to separate the collapsed lobe from the mediastinal pleura and it was easy to tie the vessels and section the lobar bronchus. Microscopic sections of the bronchus showed tuberculous lesions. All went well during the first twelve days, and the sputum became negative. The thirteenth day bronchial fistula developed, which resisted all attempts at closure. The sputum became positive again and the patient died three months later with extension of the disease to the left lung.

The second case is that of a white man, 29 years old, who had large cavities in the upper and lower lobes. Following the second stage of thoracoplasty the cavity of the upper lobe was closed, but the cavity in the lower lobe had increased in size and presented intermittent fluid level. On revision the pleural cavity was found relatively free in the lower chest and the lower lobe could be freed; the vessels were tight and the bronchus sectioned with the knife, without any crushing, carbolized, and sutured. The same complication occurred as in the previous case. Bronchial fistula was formed during the second postoperative week, which was followed by prolonged suppuration and death two months later, with signs of amyloid nephrosis.

These failures have discouraged me. However, I think that when the bronchus is already obstructed by scar tissue lobectomy may be a good procedure.

For the same reasons I do not think that lobectomy should be advised for repeated and persistent hemorrhages, when pneumothorax has failed, and before upper

*Deceased.

thoracoplasty has been performed. I have a number of such cases in which thoracoplasty was performed as an emergency operation with very gratifying results.

DR. RALPH B. BETTMAN, CHICAGO, ILL.—I am sorry that I have to report a failure too, but I think it is only fair that we report these. This is a case of tuberculosis of the upper lobe, for which it seemed advisable to do a lobectomy. The lobectomy was easily accomplished and the patient as usual made a fine convalescence until the bronchus opened up and infection occurred and she died. At the operation we noticed large matted mediastinal glands, a condition which of course is very frequently found, and I have a feeling that probably an infection could have arisen from those glands.

I would like to ask Dr. Jones whether he had any difficulty with glands around the hilus of the lung which might be the source of infection. Far from being discouraged, though, by my own failure, I still feel that there are theoretical grounds on which a lobectomy or even pneumonectomy is indicated. I am reporting this one case not to dampen the hope Dr. Jones has given us but to just be a little bit fair and perhaps add a word of caution.

DR. JOSEPH GORDON, RIDGEWOOD, N. J.—I would like to make the statement of a personal observation and suspicion I have had about these cases while working with Dr. Coryllos. We have asked many times about doing lobectomies in these cases and he always cited to us the danger of chronic suppurative wounds. We later had ample opportunity to observe some cases which made us hesitant about doing this operation. One or two of the examples have been stated.

We had occasion to operate a lung abscess at one time which later proved to be either a secondarily infected tuberculous lesion or a lesion of a tuberculous nature underlying the abscess cavity. This patient for a time went along and did fairly well after the abscess had drained; then one day, for no reason at all, he showed a positive sputum. His previous sputum examinations over a long period of time had been negative. We cultured the wound which was draining. At that time the drainage had changed in character and showed definite evidence of acid-fast infection. The patient ultimately went to his demise from this chronic suppuration. My feeling in the matter was enhanced by the new facts we are learning in doing more bronchoscopic examinations than we had done before.

In instances where we are contemplating doing lobectomies and pneumonectomies, would it be possible to assure ourselves of the fact that there are no tuberculous infections high up in the bronchial tree? I cannot see any particular reason why, with the lung removed containing the tuberculous infection, the patient should not do as well as when you remove a lung for abscess or malignant tumor. Dr. Jones has shown us this very well. I was wondering if such an observation regarding the bronchi had been made before in the series of cases Dr. Freedlander reported about two years ago. Some did well, but death was ultimately due to the chronic suppuration. It is likely that with some tuberculous tissue being left behind in the bronchus we would probably get the same sort of story as one would if one had a malignant growth and left some of it behind. It would defeat the success of the operation and the disease process would continue.

If there was no tuberculous involvement of the bronchus that was being left behind, the healing should be the same. The invading organism or the invading tumor mass has been removed and closure and accomplishments should parallel very closely.

The other thing I would like to add is a consideration of the possibility of doing lobectomies in cases where one has had a previous thoracoplasty. For quite some time we had a terrific discussion with our pathologist who told us that it was impossible to remove a lung after a thoracoplasty had been done, his experience having been gained at the post-mortem table where bleeding did not mean anything and

where he simply attempted to strip the lung away from the chest wall. Dr. Coryllos demonstrated to us that the lung can be freed under thoracoplasty without difficulty and in instances where there is a residual cavity which resists collapse it may be particularly well taken to do a lobectomy or pneumonectomy as indicated. We would perhaps get better results than we would in the instances where we do a thoracoplasty revision.

DR. ROBERT GLEN URQUHART, NORWICH, CONN.—So long as confession is good for the soul, and so that Dr. Graham shall not be fearful of any suicides, we have a case in Connecticut in which we did a pneumonectomy in which there was apparently a stricture of the bronchus. The 16-year-old girl was running a high temperature; bronchoscopic examination showed no evidence of tuberculosis, and even a Mantoux test was negative. Operation and pneumonectomy were performed and the pathologist came back to report that it was a tuberculous lesion. The patient had a persistent bronchial fistula, with a spread to the opposite side, and died five months after operation.

DR. STUART HARRINGTON, ROCHESTER, MINN.—I have recently had a case of the type which Dr. Graham mentioned, in which the diagnosis had not been established preoperatively. The preoperative diagnosis was that of bronchiectasis of the upper lobe of the right lung. An upper lobectomy was performed and the condition proved to be bronchiectasis with an associated extensive tuberculosis. The pleural cavity was closed without drainage. This patient made a very satisfactory recovery and the lung healed by primary union. There was no bronchial fistula or pleural effusion. I believe it probable that the radical removal of localized areas of tuberculosis in selected cases will be considered more frequently in the future.

DR. JOHN C. JONES, LOS ANGELES, CALIF.—I appreciate very much Dr. Graham's remarks and his review of the historical aspect.

Dr. Shipman and I agree entirely with his indication for lobectomy in the lower lobe bronchiectasis, with of course stenosis above it.

In reply to Dr. Janes and Dr. Coryllos, the year before this first case, operated for repeated hemorrhages, was done, we lost three thoracoplasties by virtue of huge hemorrhages directly after either the first or second stage of thoracoplasty. I think that if a pneumothorax is obtained, and the pneumothorax collapse about the lesion does not control the bleeding, it is a fair test that there is a great risk that thoracoplasty at the first stage will not control bleeding. As a matter of fact, in this first lobectomy the patient coughed up over 400 gm. of blood before the tourniquet could be applied about the hilar stump. If we were doing a thoracoplasty, we would not have been far enough along to have obtained compression sufficient to control the hemorrhage. I believe, then, there are indications in hemorrhage for doing lobectomy, particularly if a lung compressed by pneumothorax continues to bleed profusely.

In answer to Dr. Bettman's question, we didn't encounter any difficulty with lymph nodes in these tuberculous cases. In the last case reported where the pneumonectomy was done supposedly for carcinoma, anthracotic mediastinal lymph nodes were no problem to us.

In regard to Dr. Gordon's question, all these cases were bronchoscoped before they came to surgery. Of course in a lobectomy it is impossible to cut out all of the tuberculosis because there is infection of all the stumps of these lobes. There is no particular difficulty in freeing the lobe of the lung under a thoracoplastic collapse. I was surprised with the ease of dissection; the small space was left for the chest wall to retract and obliterate the empyema cavity.

Addendum: Since this presentation, a fifth patient, with a right total pneumonectomy, has been successfully operated, Case 5, W. L. M., 39-year-old, surgeon, re-

ferred by Dr. R. L. Cunningham. His father had died of tuberculosis at the age of 40 years. His wife and child are alive and well. Following influenza which he had while in the army in 1918 he developed pulmonary tuberculosis with a positive sputum. He had had several flare-ups of his disease since that time. In January, 1935, he became febrile, had an hemoptysis, followed by bacillary positive sputum and definite infiltration and peribronchial thickening in the right upper lobe of the lung. At no time was there roentgen evidence of cavity.

Following several months' bed rest a total right pneumothorax was induced Oct. 11, 1935. It was noted at this time that after the first two pneumothorax treatments (total 700 c.c.) by Dr. Cunningham, the right upper lobe had become entirely atelectatic and it remained so. In June, 1936, a right temporary phrenicectomy to supplement an apparently complete pneumothorax was done in an attempt to convert the persistently positive sputum. During the summer of 1936 he complained of so-called asthma and a definitely increasing wheeze. He was referred for bronchoscopy on Sept. 18, 1936. This procedure revealed tuberculous ulcerative stenosis of the right main stem bronchus which involved the distal right lateral wall of the trachea and the carina. The lumen of the right bronchus was 3 mm. in diameter.

The pneumothorax was slowly abandoned, allowing complete re-expansion of the middle and lower lobes, but the upper lobe remained totally atelectatic. The patient continued to be toxic and febrile. Deep roentgen therapy over the lesion, silver nitrate, and cautery were applied locally to the lesion through the bronchoscope, with no appreciable good effect. Tuberculin injections in small doses were not tolerated. Frequently recurring febrile episodes terminated by the expectoration of increasing amounts of retained, purulent and bitter-tasting sputum, with increasing toxicity of the patient, led us to believe that pulmonary suppuration distal to the stenosed main stem bronchus was the cause of his downhill course. Since the bronchus was almost closed off, bronchography was not attempted. In September and October, 1937, a three-stage seven-rib Semb thoracoplasty was completed preparatory to pneumonectomy. Bronchoscopy, May 6, 1938, revealed complete occlusion of the right main stem bronchus with granulations which were cauterized and an opening established. His general condition had only slightly improved since thoracoplasty. On May 30, 1938, under cyclopropane anesthesia, a total right tourniquet pneumonectomy was done through a curved periscapular incision from the fourth interspace posteriorly to the seventh interspace in the midaxilla. There was considerable soiling due to the escape of copious amounts of pus as the bronchi were cut across during the amputation of the lung. The wound was loosely packed with gauze saturated with 40 per cent zinc peroxide to allow good drainage and, at the same time, to steady and support the extremely mobile mediastinum. Five hundred c.c. of whole blood were administered at the close of the procedure although there was no evidence of shock and there had been a minimum of blood loss.

The resected lung was that of extensive bronchiectasis in all three lobes. There was considerable thickening of the bronchi, grossly multiple small tuberculous caseating areas, and direct smears of the lung were loaded with tubercle bacilli. The middle lobe showed considerable pneumonitis. There were no gross tuberculous cavities noted.

The patient's postoperative convalescence was entirely without event. A few weeks postoperatively he had transient extrasystoles which were controlled by quinidine. The wound has shown progressive healing although there persists a draining sinus at this time. Cauterization of the stenosing bronchus through the bronchoscope was carried out Sept. 30, 1938, and Nov. 4, 1938. Bronchoscopy on Feb. 10, 1939, revealed a completely stenosed and entirely healed-over lesion of the bronchus. The small amounts of mucoid sputum are negative for tubercle bacilli on smears and concentrations. The patient has shown progressive improvement, is afebrile, and his left lung has remained entirely clear according to roentgen and physical examination.

THE FORMATION AND DEVELOPMENT OF THE PRIMARY TUBERCLE IN THE LUNG*

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THE reaction of virgin pulmonary tissue to invasion by the bacillus of tuberculosis creates a lesion known as the primary complex that is made up of three anatomic entities, represented by the tubercle that develops in lymphoid tissue at the portal of entry, the lymphatic channels that drain it, and the regional lymph nodes which are interposed as more or less ineffectual filters or "sedimentation basins"[1] between the primary focus and the general circulation. Cummins conceives of the three affected parts as constituents of a unit in which "lesions of primary localization" may occur simultaneously following entrance of the bacillus into a lung which has not been made sensitive by previous tuberculous infection.

These lesions are distinctive in that they usually represent a simple reaction to a very few bacilli, which we believe are most often brought directly into the lung during respiration. Calmette thinks differently. He says: "I myself affirmed, with Ravenel, Aufrecht, Klebs, and many other investigators, that in all susceptible animals, man included, tuberculosis, in all its various localizations, glandular, pulmonary, and so forth, particularly in its slowly evolutive forms, results in an immense majority of cases, from an infection which is primarily lymphatic and later of the blood, and which originates in the absorption of tubercle bacilli from the digestive tract, principally through the buccal, pharyngeal and intestinal mucous membranes."

In the human body it is impossible to know the sources of infection or the time when infection takes place, and it is impossible to follow the first cellular reactions that occur at the point of invasion or during migration of bacilli to the point of arrest in the pulmonary tissue or during their passage out of the lung by way of lymphatic vessels or the bronchial system. These preliminary reactions can be studied only by experimental methods. The inhalation method used by Baldwin and others is best suited because the results simulate tuberculosis most closely in nature. By this method, small numbers of bacilli are scattered widely throughout the lung and initiate lesions but cause minimal trauma and reaction to foreign protein. Griffith employs a similar method, making use of the Buchner apparatus.

*Read (by title) before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery, Atlanta, Ga., April 4, 5, and 6, 1938.

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The description of these observers records the first responses, of microscopic size, elicited in segregated regions in the normal lung, in the regional lymphatic tissue, and in the associated lymph nodes. By such methods, the earliest cellular changes may be studied microscopically as they are developed in the primary complex. The progress of development and the final state of the primary complex can be studied only by a combination of experimental, clinical, and pathologic investigation. Study of the developing complex necessarily must include the reactions of both unsensitized and sensitized tissue to the specific organism, because the lesion it creates starts in a lung unchanged by previous contact with the bacillus or its products. However, before metamorphosis in the primary complex is complete, the pulmonary tissues are sensitized and the nature and composition of the lesion are influenced by complex allergic reactions consequent to autogenous reinfections.

In this account I desire to record my observations of the development of the primary tubercle and its associated lesions, by both experimental and clinical methods.

METHODS OF EXPERIMENTATION

In the course of my observations I have made use of all the standard methods of infecting the lungs of normal animals with the bacillus of tuberculosis. The infection produced by each method effects distinctive pathologic changes of which the histologic tubercle is pathognomonic. By intravenous injection, the lungs become filled with miliary tubercles, evenly distributed; by subcutaneous injection of bacilli, the result is almost identical because the bacteria are carried by lymphatics into the circulating blood, and that which started as a subcutaneous inoculation has the same effect as an intravenous inoculation. By intratracheal introduction, the lesions obtained are more complex because of the massive dosage of bacteria, the traumatic effect of the inoculum, and the temporary atelectasis which occurs soon after inoculation. The lesions induced are more nonnodular than nodular in form, less discrete and more diffuse than miliary in character. The great tendency of tubercles to become conglomerate is observed best when the intratracheal method is used. Also, the immediate necrotizing action of the bacillus or of its constituent fractions set free by death of the bacillus or of the enzymes released by the disintegration of polymorphonuclear leucocytes is best seen when the intratracheal inoculum contains vast numbers of organisms of high virulence. Within one week following inoculation, necrotic alveolitis may be studied. As previously stated, the inhalation method produces a result which nearly simulates such infection among human beings. But, regardless of the method of introduction, development and dispersion of lesions soon become identical, and each method always affords the opportunity for observing the very earliest reactions.

The first microscopic tubercle, regardless of the method of introduction, usually develops where lymphoid tissue is normally present. "In experimental tuberculosis, the law of Cohnheim holds good—at whatever point the tuberculous virus is introduced or penetrates an epithelial surface, there, or in the adjacent lymphatic glands, a tuberculous lesion appears."[4] Thus, the primary localization is determined by the portal of entry and, among susceptible animals, the primary localization of lesions always indicates the avenue of infection. Cobbett said that by inhalation particles of great fineness are drawn into alveoli and out to marginal portions of the lung within a few minutes. By inhalation alone, 21.5 per cent of moist spray and 12 per cent of dry dust reaches the alveoli. I found that bacilli could be detected in subpleural parenchymatous tissue almost immediately following intratracheal injection of bacilli suspended in a normal salt solution.

REACTIONS BROUGHT ABOUT BY THE BACILLI

The series of reactions which ultimately brings about formation of the first or primary tubercle begins when the bacteria become intracellular in position within the peripheral alveoli. The first of the reactions of the pulmonary tissue to contact with the organism is typically inflammatory and in no way differs from those reactions resulting from presence of any other insoluble or refractory particulate irritant of similar size and indestructibility. The bacillus varies in size, but the average is estimated to lie between 1.23 and 4.12 microns.[6] The most dangerous dusts of industry are composed of particles varying from 1 to 3 microns in diameter. It may be said, however, that the bacillus or its waxy envelope causes less irritation than many dusts that are similarly carried into the peripheral alveoli. For this reason inflammation is inconspicuous and transient. Yet capillaries in alveolar walls are dilated and conspicuous, serum can be detected in alveolar spaces, and there is an escape of erythrocytes and polymorphonuclear and mononuclear leucocytes from the circulating blood. The alveolar phagocytes appear enlarged and are shed into the alveolus where they collide with bacilli. Within a period of six hours it is difficult to detect an extracellular bacillus, so quickly does phagocytosis occur. It is easier to demonstrate this observation by use of particulate graphite, the color of which allows easy identification. It is legitimate to do so because the pulmonary cells react toward graphite and phagocytose its particles in precisely the same way as they do the wax-covered tubercle bacillus.

The three factors requisite for phagocytosis are present in the lung which is mildly inflamed by the presence of bacilli or of graphite, namely, serum, phagocyte and foreign body. The chance for collision between the phagocyte and the particle of graphite or the bacillus is the same whether contact is purely fortuitous or by virtue of locomotion of the phagocytic cell. Moreover, in the presence of either the graphite or the bacillus, phagocytosis often is continued until the phagocyte is filled to the point of rupturing.

The similar reaction of all phagocytic cells induced by the presence of particles of graphite and bacilli of tuberculosis is not caused by all foreign substances, however. Phagocytic cells appear to discriminate between certain substances; they may actively phagocytose some and completely reject others. Mudd and Fenn have observed the same difference in behavior. Although there may be no differences in the chance of collision, in our experience, the polymorphonuclear cell rarely ingested silica, whereas either graphite or bacilli were phagocytosed with great avidity.

Feldman and I have been able to find no difference between the behavior of minute inorganic particles and bacilli following their deposition in pulmonary alveoli, during their ingestion by phagocytes, or in the course taken as they are carried out of the lung.

During the active period of phagocytosis and afterward, the polymorphonuclear and mononuclear cells and the alveolar phagocytes wander through the interalveolar partitions. There are no lymphatics in alveolar walls and movement must be largely ameboid.[10] They may be carried passively by intra-alveolar fluids, which are present as a result of irritation induced by the presence of the particles, toward the distal openings of the alveolar ducts. Compression of the lung by expiration also may help carry the phagocytes to the duct where, after a few hours, masses of cells can be found. Sometimes, smaller concentrations of cells may be seen held in the tiny masses of lymphoid tissue that may be detected at the distal end of the ducts.[10] Their identification is made easy owing to the color of the graphite or use of a specific stain when bacilli are being searched for. Within twelve hours, phagocytic cells laden with bacilli can be identified in subpleural, perivascular and peribronchial situations, carried there by the superficial and deep lymphatics having origin at the distal ends of the alveolar ducts. These laden phagocytes may be seen also as constituents of bronchial exudate as it proceeds toward its exit from the airway. At the same time, similar cells can be identified in the regional tracheobronchial lymph nodes. Tuberculous lesions develop wherever intracellular bacteria are deposited and impounded in the course of their journey from the pulmonary parenchyma to the regional lymph node.

As MacCallum has said, "It would appear that the distribution of coal pigment in the lung must offer a model upon which we could reconstruct the distribution of the bacilli." But, thereafter, the behavior of the tissues toward graphite and bacilli differs greatly. The inorganic substances are irritants only and the reaction to them is relatively uncomplicated; the lung treats all inorganic substances by a common mechanism of defense. It dissolves, destroys, segregates, or removes them. But the bacillus is a living particle, capable of increasing numerically and thereby changing reactions because of the increasing number of particles involved and because of the variations in the amount

of tuberculoprotein that comes in contact with living cells. Moreover, the bacillus may be killed by its phagocytic host and, as it degenerates, it may liberate stimulating substances that, for want of a more exact term, have been called toxins. Also, death and disintegration of the phagocytic cells, especially of the polymorphonuclear leucocytes, may cause liberation of enzymes that influence cellular activity.

It is the belief of competent observers that ferments of cells may cause proliferation of phagocytic mononuclear cells. Be that as it may, wherever there are bacilli, proliferation of mononuclear cells will be observed and the cellular hyperplasia rapidly becomes much more conspicuous and dominant and replaces the initial, transient, exudative phenomena induced immediately subsequent to implantation of the bacilli in the peripheral portions of the lung. Initial exudative phenomena, ameboid wandering, mitosis, and mononuclear proliferation with arrest in lymphoid tissue, constitute the essential characteristics of the earliest response of tissue to infection by the Mycobacterium tuberculosis.

The subsequent history of the tiny concentrations of cells that have been formed owing to the response of tissue to a very few bacilli can be followed by necropsy of experimental animals at various intervals of time after the bacilli are placed in the lung. Within ten hours the relationship between the numbers of the three chief varieties of phagocytic cells present alters appreciably; the polymorphonuclear cells become fewer and the mononuclear phagocytes are increasing rapidly in number. That true proliferation is occurring can be established by finding mitotic figures, even at so early an examination. After twenty-four hours have passed, most of the cells of the exudative type have disappeared, the polymorphonuclear leucocytes are fragmented, their cellular limiting membranes are destroyed, and their contained bacilli are liberated. The mononuclear cell increases both in numbers and in importance as a phagocyte. If the number of virulent organisms is great, there may be evidence of interstitial mononuclear alveolitis and obliteration and deformity of alveolar spaces surrounding the perivascular and peribronchial concentration of phagocytic cells. If the numbers of bacilli are few and their virulence is attenuated, the pulmonary spherical focus is discrete, is sharply demarked from the surrounding normal parenchyma, and is entirely cellular. Mononuclear cells predominate, but polymorphonuclear cells and a few lymphocytes may be found at the periphery of the spheroid. Within three to seven days necrotic changes occur in the center of the tiny nodule and progress to include, also, the peripheral cells. Fatty degeneration occurs, as it does when the phagocytic cells hold certain nonbacterial particles, nuclei stain more deeply than normally, fragmentation appears, and the outline of cells becomes indistinct or indistinguishable. Even in these very young lesions of microscopic size, the central cells are necrotic and are encir-

cled by a layer of dying cells. When stained with Congo red these appear red owing to the presence of fat.

Before the end of two weeks following the introduction of bacilli, the mononuclear cells of the tubercle increase in number, enlarge, and arrange themselves radially in solid cellular concentrations. We believe these cells, now classified as epithelioid cells, are derived from the original phagocytic mononuclear and alveolar cells by metamorphosis. At this time, giant cells appear. Around the central core of degenerated cells and encircling lamella of epithelioid cells, a mantle of granulation tissue develops in which layers of fibrous tissue are formed as a result of hyperplasia of fibroblasts. It is believed generally that the fibroblasts are derived, at least in part, from the adjacent connective tissue normally present.

As a result of our observations on the development of the silicotic nodule, it seems possible that the mononuclear cells developed by metamorphosis into fibroblasts or cells resembling fibroblasts and contributed to formation of a fibrous envelope about the cellular nodule. Lymphocytes are dispersed among the cells that make up the layers of granulation tissue. Compression of the developing tubercle arranges these layers in an encircling and concentric manner so that the whole mass of cells assumes an onion-skin arrangement. Fibroblasts penetrate from the periphery of the nodule and pass toward the central necrotic core, and after four or five weeks interlacing strands of collagen are laid down and often fill the whole tubercle. The tubercle itself is entirely avascular, as MacCallum has demonstrated, but newly formed blood vessels frequently are seen in the envelope of granulation tissue. Blood vessels often can be demonstrated better in the silicotic nodule than in the tuberculous nodule.[12] Whether the tubercle is obliterated and its further activity is strangled, or becomes a focus for dissemination, depends entirely on the balance between establishment of fibrosis on the one hand, which, proceeding from without, may replace the tubercle completely and segregate the bacilli effectively, and caseation and necrosis on the other hand, which, progressing from the central core toward the periphery, may break through the concentric layers of the tubercle and effect release of bacteria, causing locally the development of daughter tubercles or, by continuity of tissue or by metastasis, creating progressive disease.

Establishment of the anatomic tubercle invested with granulation tissue and composed of a necrotic core, giant cells, bacilli, epithelioid cells, and fibrous tissue represents the primary and pathognomonic reaction of virgin pulmonary tissue to the Mycobacterium tuberculosis. The tuberculous lesions of the lymphatic system of vessels and regional nodes, combined with the pulmonary tubercle, constitute the primary complex of tuberculosis.

THE PRIMARY INFECTION OF LYMPHATICS AND LYMPH NODES

The development of tuberculous lesions in the lymphatics that drain the primary focus and have their source in the regional lymph nodes is less easily demonstrated than that of like lesions in pulmonary tissue itself. Many times, I have seen spheroid lesions caused by industrial dusts in the course of the superficial lymphatics of experimental animals and have observed mononuclear cells crowding and obstructing the large vessels that make up the plexus of the deep lymphatic system. In the same way cells which contain bacilli of tuberculosis may become lodged. These produce, along the superficial lymphatics in the interlobular partitions, tubercles in strings or clusters that give the same appearance of beading that often has been observed in lesions of silicosis. Thrombosis of vessels frequently is seen affecting the arteries and veins and it is seen, although less frequently, in the lymphatics. Davis has demonstrated convincingly all stages of thrombosis and the progressive changes from canalization to obliteration in the pathologic material he studied at the Mayo Clinic. Both nodular and nonnodular or diffuse changes in tissue may be produced and the lumen of the lymphatic vessel may be completely or partially occluded. The pleural vessels become dilated, tortuous, and thickened, and may be readily seen. Their thickened walls, resulting from proliferative changes of a nonnodular type, may be beaded owing to the presence of tubercles in which the same changes occur as take place in the tubercle formed in the lung. Healing, as elsewhere, takes place through hyaline changes and scarring. In late stages, the lymphatic vessel becomes an obliterated, fibrous cord, and the tubercles in its wall become fibrous or calcified nodules. I have seen one instance, in the lung of an aged person, in which both calcareous and bony replacement occurred which obliterated the lymphatics.

In the third constituent of the primary complex (the tracheobronchial node), tuberculous lesions appear within two or three weeks following inoculation. The bacilli are lodged in the sinuses or in the lymphatic cords and there tubercles develop. These may remain discrete, but, usually, they exhibit the common trait of coalescence so that the substance of the node is filled with conglomerate tubercles. Caseation may transform the node into a cheesy mass enclosed in fibrous tissue. The node may be changed into a stony body created by deposition of coarse calcareous granules. Even in the very densely fibrous nodes, the conglomerate nature of the lesion is revealed by the presence of circular bands of fibers that surround the individual tubercles and set them apart. Susceptible animals and young infants do not live long enough for either calcareous or bony replacement to occur. Caseation is the dominating condition of the lymph nodes of such subjects.

THE NATURE OF RESPONSE OF TISSUE TO INFECTION

It has been agreed by most observers that production of the primary tubercle is a foreign body reaction. Certainly all the progressive steps in its development are similar to those that occur when silica, rather than the bacillus, is the irritant and, again, with minor exceptions, when organisms responsible for infectious granulomatous diseases such as syphilis, brucellosis, and tularemia react with pulmonary tissue. However, there are many who consider tuberculosis as an infectious disease different from all others, and they offer convincing reasons for thinking that some specific influences determine the nature and form of its lesions. Dienes and Mallory think that sensitization of cells from contact with the organism makes allergic reactions possible even though the interval of time is brief. They think that allergy may play a fundamental role in the pathogenesis of the disease and that, in part at least, it may determine the character of the anatomic lesions and, especially, the predominance of large mononuclear phagocytes. From their own experiments and from those of Stewart they conclude that allergic reactions may occur with the appearance "of significant numbers of large mononuclear cells." They report the occurrence of demonstrable sensitivity to tuberculin on the third or fourth day after primary infection.

There is a variety of other explanations for cellular proliferation, fatty degeneration, and necrosis of the cells constituting the earliest concentration of phagocytes. We have been of the opinion that the bacillus or its waxy envelope or the chemical fractions released by death and disintegration of the organism are sufficiently active irritants to be responsible for the necrosis of the phagocytic cells that are present during the first week following primary infection. Similar degenerative changes occur with many irritants: sericite, asbestos, and other silicates frequently kill the phagocytic cells that enclose their particles. It is a reasonable hypothesis that phenomena of proliferation and coagulation necrosis represent purely nonspecific reactions of cells to irritation and are common to many organic and inorganic particles.

The ferment derived from the dead polymorphonuclear cells presumably may contribute to cellular degeneration. Other observers have assumed that the bacilli of tuberculosis produce "toxins" and that the cellular hyperplasia and degeneration are end results of the action of toxins. Griffith says: "The toxin of the living tubercle bacillus is a protoplasmic irritant poison and, according to its potency and concentration and the kind of cell involved, either causes coagulation necrosis of the protoplasm and death of the cell, or exerts an irritant action leading to cellular proliferation, usually a combination, and relative amounts of the two effects determine the character of the focus: (1) caseonecrotic and destructive; (2) formative and neoplastic."

Further support of the hypothesis of the production of toxins is given by Davies who believes that, with arrest and survival of the tubercle

bacillus, the phagocytic cell containing the organism degenerates and liberates toxins which cause proliferation of cells epithelial in appearance. He also suggests that the avascularity of the granuloma and, to a degree, the toxins liberated from the bacilli, bring about the characteristic caseation in the center of the tubercle. Caseation, he thinks, is a process of fatty degeneration resulting in formation of a cheesy mass. Proteolytic digestion of the caseous content is believed responsible for its transformation into a thin liquid containing split proteins, lipoids, salts, and acids.

The nature of the response of tissue, therefore, is influenced presumably by the irritating quality of the bacillus or by the products resulting from its death or by the products of disintegration of polymorphonuclear phagocytes; these, in turn, are influenced by development of sensitization of cells and allergic responses to antigens.

Before accepting any of the theories advanced it is well to remember that there should be more enzymes released from polymorphonuclear cells in cases of experimental silicosis, where polymorphonuclears are found in larger numbers, than in tuberculosis; yet, less necrosis is associated with silicosis than with tuberculosis. Again, as far as can be determined, toxins such as those referred to have not been isolated, and, as Krause remarks, the assumption that an endotoxin exists is poor support for the theory. He says: "'Endotoxins' as inherently specific poisons must be regarded as purely hypothetical constituents of a microorganism; and in the circumstances 'endotoxins' have never enjoyed any more real existence than the designation of them as an imagined substance to account for an effect observed." Regarding the question of allergy raised by Dienes and Mallory, our experience leads us to believe that sensitization of cells begins soon after contact with the bacillus, that it increases with the passage of time, and that, usually, it can be demonstrated within two or three weeks, as soon as sensitization is sufficiently developed to produce inflammation on reinfection by specific organisms. There seems little doubt that the focal allergic manifestations of secondary infection are not different, but merely intensified reactions frequently produced at first contact of tissue with tubercle bacilli or other irritants. Of all the irritants mentioned, the bacillus is the only one that contains a sensitizing protein and that is capable of increasing in numbers and changing in virulence. It is, therefore, the only irritant capable of causing sensitization or intensifying sensitization so that an altered reaction of violent type may be brought about. Proliferation, necrosis, and fibrosis may be reactions that all irritants, in common, may produce, but sensitization allows of uncommonly intensified reactions that make them seem to be specific rather than nonspecific. If focal cellular sensitization can be developed to a sufficiently high degree within three to five days, it may well be that local allergy occurs much earlier than can be demonstrated by

reactions to tuberculin, and, therefore, proliferation and the first detectable signs of cellular necrosis are due to allergy. Certainly, necrosis is associated more conspicuously with tuberculosis than with silicosis or other types of granuloma and is of a different order; also, the end result, caseation, is peculiar to tuberculosis. The disproportion between the numbers of bacilli within the tubercle and the amount of necrotic tissue present lends support to the hypothesis suggested by Dienes and Mallory. The earliest demonstrable reaction to tuberculin obtained by Montgomery and me occurred on the fifteenth day, that is, a week following detection of the first immature histologic tubercle. Thereafter, pneumonia, necrosis, ulcerative phenomena, thrombosis of vessels, and effusion dominated the pathologic changes, and constitutional evidence of illness became apparent. These are the changes that occur when susceptible infants or people of primitive races succumb to infection by a virulent, human strain of bacilli. It is not the usual sequence of events in what is classified as primary tuberculosis, for primary tuberculosis almost invariably does not produce symptoms and proceeds toward curing rather than toward disseminating the disease.

ADDITIONAL NOTES ON DEVELOPMENT OF THE PRIMARY TUBERCLE

The primary tubercle consists of a lesion of microscopic size. This small collection of cells increases in size by proliferation and by the formation and coalescence of daughter tubercles (demonstrated so well in study of tuberculous lesions of fowl), by coalescence of neighboring tubercles, and by extension locally and establishment of tuberculous alveolitis and pneumonitis. The initial focus is, thus, a localized pneumonitis. Its subsequent development and change in constitution are best followed by clinical and pathologic examination. Two years, at least, may pass before this slowly developing innocuous pneumonic lesion becomes obliterated by fibrosis, calcification, or before bony replacement and sterilization occur owing to death of the contained organisms.

Baldwin, Petroff, and Gardner believe that the caseous material at the center of the tubercle may become calcareous owing to deposition of lime salts derived from the blood. These salts, consisting of magnesium phosphate and calcium carbonate, exist in such lesions in the same relative proportion as they do in normal bone. They state, also, that when living fibroblasts penetrate caseous material, ossification may occur instead of calcification.

Neither calcification nor ossification of a tubercle ever has been observed by us on examination of infants or experimental animals because, as a rule, their disease is progressive and ends fatally too soon to permit occurrence of such healing processes. Experimentally, these changes have been observed by others. Both have been observed frequently in human material obtained at necropsy. Radiologically, the pulmonary lesions among adults are usually single, sometimes multiple, but may

appear as large numbers of discrete nodules surrounded by normal pulmonary tissue. They are more basal in situation than apical and more often are situated peripherally in the lung rather than centrally. They vary in size from very small, circular, opaque shadows 1 to 2 mm. in diameter, to masses irregularly spherical in shape, measuring from 1 to 3 cm. in diameter. Evidence of lymphatics occasionally can be found on roentgenologic examination, and the regional tracheobronchial lymph nodes give evidence of enlargement and contain islands of opacity owing to the deposits of calcium in them. If the pulmonary focus is calcified, the lymph nodes belonging to the drainage system, likewise, are calcified.

The conglomerate pulmonary, lymphatic, and glandular lesions that have become replaced successively by fibrous tissue or by caseous material and have become calcified or ossified, represent the final state of a tuberculous process which, starting in previously uninfected tissue, develops slowly as an indolent, innocuous lesion and, before reaching maturity, brings about local and then general sensitization of the cells of the lesion itself and of all the tissues of its host. Long after establishment of sensitization, sufficient to be revealed by the tuberculin reaction, autogenous reinfection is possible and takes place because living virulent organisms are shed from the pneumonic focus for a long period of time.[20] The interval of time frequently permits development of those secondary infections of the pleura and of the pulmonary tissue at the apex of the lung. Theoretically, such reinfections should be met by allergic, clinical reactions of a highly inflammatory character. Very often, symptoms of illness are not produced at all, or are so mild as to be unnoticed. There must be a fine balance between immunity and allergy during development of the primary complex, not only to prevent the spread of active disease, but also to make the lesion of no immediate clinical importance.

HEALING PROCESSES IN THE LYMPHATICS AND TRACHEOBRONCHIAL NODES

At the time of establishment of the first pulmonary tubercle, similar changes always are found in the lymph node that receives the drainage from the affected region in the lung. As previously stated, Davis has shown that similar changes have occurred along the lymphatics that carry the lymph and intracellular bacilli from the primary focus to the lymph node. MacCallum has illustrated the beading along lymphatic vessels created by the development of tubercles. We have seen similar nodules produced by the action of silica and have seen lymphatics of the tuberculous lung of human beings obliterated by calcification. We have found tubercle bacilli in the tracheobronchial lymph nodes twelve hours after intrapleural injection of bacilli. Cobbett says that the bronchial glands contain bacilli five days after infection by inhalation. They are enlarged by the twelfth day and contain lesions by the twenty-third day. The pulmonary lesions he speaks of were visible by

naked eye in nine days. Once established in the lymph nodes, the lesions have the same marked tendency to become confluent as they have in the lung. Caseation, fibrosis, calcification, and ossification proceed in a parallel manner. There is the same tendency to develop slowly and to be innocuous and there is the same difficulty in attempting to discover them other than by use of roentgenologic and pathologic methods.

Occasionally, although rarely, the lymph nodes act as very inefficient filters and such large numbers of bacilli drain into the circulating blood that they cannot be destroyed, and miliary disease results. Rarely, also, we have seen the nodes become so large that they assume the proportions of a tumor; thus, they exert pressure on vessels and nerves in that region and bring about establishment of superficial collateral venous circulation, the syndrome of Horner, paralysis of the recurrent laryngeal nerve, cough, dyspnea, and stridor. Less rarely, they may exert pressure sufficient to ulcerate through a large vessel and induce hemorrhage or miliary dissemination, or invade the lumen of a bronchus and produce tuberculous pneumonia, or, as a lung stone, induce stenosis and secondary bronchiectasis or pulmonary abscess.

PROGRESSIVE LESIONS IN TRACHEOBRONCHIAL LYMPH NODES

Pinner and Kasper have described the progressive changes that occur in lungs and in lymph nodes of Negroes. Recently, Auerbach described a progressive type of primary complex seen at necropsy of a series of sixteen Negroes and one white child. The ages of these children ranged from 9 months to 9 years. The highest incidence of progressive lesions was found in the age period in which the greatest number of primary foci develop, that is, fifteen cases occurred between the ages of 1 and 3 years. Auerbach found that progression and softening occurred simultaneously or independently in both primary pulmonary focus and draining lymph nodes. In two of his cases, a caseous node had ruptured into a neighboring bronchus.

However, Negroes and very young children may react to their first infection in the same manner as experimental animals. The primary complex in them is then progressive, the lesions are disseminated and are characterized by allergic phenomena represented by pneumonia, pleural effusion, necrosis, and ulceration into neighboring bronchi or blood vessels.

In contrast, the initial tuberculous infection of less susceptible persons occurs at the portal of entry and is developed from retention of few bacilli wherever lymphoid tissue normally occurs. Together with its lymphatics and its draining lymph nodes the lesion passes through the various changes of pneumonitis, caseation, fibrosis, calcification, or ossification. The bacilli first inhaled or brought to the lung through the circulation, as well as those that were added in the course of develop-

ment of the lesion, are so completely encapsulated that they die. Feldman, Baggenstoss, and others have shown that in the final stage of development of the primary complex the lesions are sterile. The threefold lesion involving the lung, its lymphatics, and regional lymph nodes is always the first to be developed, is usually indolent in its growth, usually is of itself harmless, and represents the oldest tuberculous lesion in the body.

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BRONCHOPLEURAL FISTULAS COMPLICATING PULMONARY TUBERCULOSIS

A Clinical Pathologic Study

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THE occurrence of bronchopleural fistulas in pulmonary tuberculosis is a well-known phenomenon and constitutes one of the gravest complications of that disease. The presence of an ulcerative communication between the bronchial tree and the pleural space leads not only to the presence of an excess accumulation of air in the pleural space, but also to an influx of tubercle bacilli along with various pyogenic organisms. The latter in turn lead to the formation of a mixed tuberculous and pyogenic empyema. Aside from the severe toxemia produced by such accumulations of pus in the pleural space, there is also always the danger of a spill of the pus through the fistula into the lung parenchyma causing new and further progression of disease. Although tuberculous patients may live a number of years with such fistulas, usually the formation of such fistulas is of gravest prognostic importance, and unless quickly diagnosed and adequately treated, the life expectancy in such cases is very short.

With the extensive tuberculosis autopsy material available at Sea View Hospital, bronchopleural fistulas are encountered with relative frequency and since a review of the literature concerning the incidence, pathogenesis, and pathology revealed an extreme paucity of data, it was decided to collect and review clinically and pathologically all such cases in which autopsy protocols were available.

GENERAL CONSIDERATIONS

In 1000 consecutive autopsies of chronic ulcerative pulmonary tuberculosis, bronchopleural fistulas occurred in seventy-eight cases (7.8 per cent).

Age.—The age of the patients who died of bronchopleural fistulas ranged from 8 to 64 years, the greatest incidence occurring between the age of 20 and 40 years. This corresponds to the mortality incidence in chronic pulmonary tuberculosis (Fig. 1).

Sex.—In this series of cases there were fifty males and twenty-eight females. This is in direct proportion to the number of male and female patients who come to autopsy.

Color.—Of the seventy-eight cases, forty-seven occurred in white patients and thirty-one in negroes. This corresponds to the ratio of colored

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Read before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

to white patients who come to autopsy. It has been stated that tuberculosis in the negro has a greater tendency to ulcerate and is of predominantly exudative character, and one would expect to find a greater number of fistulas. This, however, was not the case.

Number of Fistulas.—In sixty-seven cases in this series a single fistula was found. In the remaining eleven cases multiple fistulas were observed, four being the greatest number of openings found in any case.

Size.—The size of the opening in the visceral pleura may vary from 1 mm. to 4 cm. The larger openings are present in those cases in which an artificial pneumothorax had previously been induced.

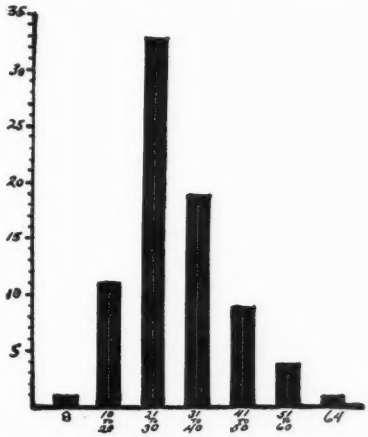

Fig. 1.—Distribution of age groups of patients with bronchopleural fistulas present at autopsy. Numbers at the left represent number of cases; numbers at bottom, age in years.

Location.—Of the seventy-eight cases of bronchopleural fistula, fifty-seven instances of upper lobe openings were found as compared with eighteen in the lower lobes. In three instances fistulas were found in the middle lobe. This fact is not surprising since the fistulas arise from the perforation of a liquefying tuberculous process through the visceral pleura, and it is well known that tuberculous processes are far more frequent in the upper than in the lower lobes.

The bronchopleural fistulas observed at autopsy can be classified into three distinct groups: (1) fistulas occurring in those cases of pulmonary tuberculosis in which artificial collapse of the lungs has never been present, (2) fistulas occurring in lungs which have been compressed by artificial pneumothorax, and (3) fistulas occurring as a direct result of closed pneumonolyses (Jacobaeus) operations.

1. Bronchopleural Fistulas Occurring in Previously Uncollapsed Lungs

The occurrence of fistulas in this group of cases is usually associated with, and the immediate result of, an overwhelming, acute caseous pneu-

monic process. The patient as a rule dies soon after the formation of such fistulas and this gives an excellent opportunity to study the early pathologic findings.

There were fifteen cases (19 per cent) found at autopsy which could be placed in this group (Table I). All the patients were between 24 and 30 years of age with the exception of three who were 45, 53, and 64 years old. Eleven of the cases occurred in white individuals and the remaining four in negroes. In only five instances were clinical diagnoses made of the presence of the fistulas. In two instances the diagnosis was made by the presence of a sudden massive pneumothorax shortly before death. Three patients entered the hospital with fistulas already developed, two of which were also complicated by the presence of silicosis. All the remaining patients had fulminating caseous pneumonic tuberculosis with rapid downhill courses followed by death.

TABLE I

DATA OF AUTOPSY CASES OF BRONCHOPLEURAL FISTULAS OCCURRING IN THE ABSENCE OF A PREVIOUS ARTIFICIAL PNEUMOTHORAX

Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Age	30	24	32	32	25	24	53	38	45	36	31	25	64	30	29
Sex	M	F	M	M	M	F	M	M	M	M	F	F	F	F	M
Color	N	W	W	W	W	W	W	W	W	W	N	W	N	W	N
Duration of illness (mo.)	6	18	6	24	24	24	36	24	12	24	12	24	3	12	6
Clinical diagnosis	Y	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	Y
Methylene blue	*NI	NI	NI	NI	NI	NI	E	E	NI	NI	NI	NI	NE	NI	NI
Size of fistulas (mm.)	S	40	L	?	10	10	?	?	S	S	10	?	2	?	2
Location of fistulas	LL	RM	RU	RU	LU	LU	LU	LL	LL	LL	RU	RL	LU	LU	RU
Number of fistulas	1	1	1	1	1	1	1	1	1	2	1	1	1	1	2

*Abbreviations: NI, not instilled; NE, not expectorated; E, expectorated.

There are a number of interesting observations in this group. From Table I it can be seen that the proportion of negroes to white individuals was four to eleven. This is somewhat unexpected since one would expect the fulminating type of pulmonary tuberculosis to be more common in the negro group (Pinner and Kasper[1]).

It is not surprising to find that in only five instances the bronchopleural fistulas were diagnosed clinically. These patients usually are admitted to the hospital with a far-advanced rapidly spreading disease and are critically ill. As a result very few or no diagnostic measures can be instituted. Thus methylene blue was injected in only three cases and expectorated in two of these. The duration of illness in this group was relatively short, varying from three months to one case with a three-year history. The average duration of the disease was from one to two years.

Pathology and Pathogenesis.—Bronchopleural fistulas in this type of involvement may vary in size from small openings to very large ones (4 cm.). They lead directly into tuberculous excavations situated just beneath the visceral pleura. These excavations are usually small, and of recent origin as shown by the soft, caseous material present in them and the small degree of productive reaction in their walls. The edges of the visceral pleura bordering the fistulas are usually caseous and the surrounding visceral pleura is covered with fibrinous exudate.

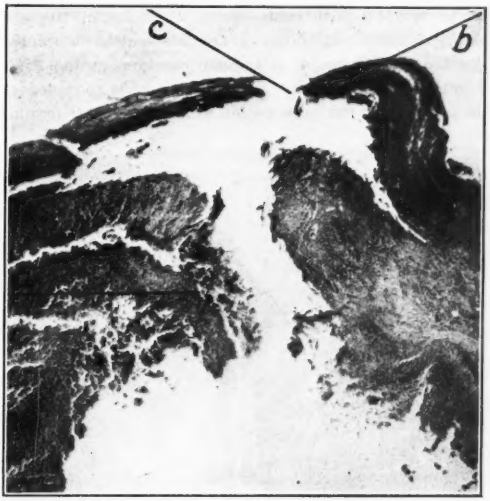

Fig. 2.—Photomicrograph through the site of a spontaneous pneumothorax. The caseation of the lung tissue (*a*) has extended to involve the visceral pleura (*b*). Liquefaction of the caseous process has resulted in a small perforation of the visceral pleura (*c*).

The origin of the perforation is a subpleural area of caseous lobular pneumonia which has extended to the pleura, the latter also undergoing caseation (Fig. 2). When liquefaction takes place within the caseous pneumonic zone it extends to involve the pleura, so that the cavity which develops communicates with the bronchus as well as with the pleural space. The parietal pleura in such cases is only slightly thickened, and present within the pleural space is a small amount of fluid and fibrinous exudate containing acid-fast bacilli and rarely pyogenic organisms. The larger the opening, the more extensive is the exudate present. The lung containing the perforation is greatly compressed and when the chest is opened is seen to lie close to the vertebral column.

The formation of fistulas in untreated cases as described above is a relatively rare occurrence. The reason for this is that in the great majority of cases the caseous foci within the lung do not reach the visceral pleura to involve it in the caseous process. Most often a zone of perifocal inflammation lies between the visceral pleura and the caseous focus.

This perifocal inflammatory reaction between the subpleural caseous foci and the pleura becomes organized and leads to fibrosis, thickening, and fusion of the visceral pleura and parietal pleura. The fused and thickened pleuras cause complete obliteration of the pleural space and also form a resistant barrier to the tuberculous process within the lung.

REPRESENTATIVE CASE REPORT

CASE 1.—V. P., a 31-year-old negress, was admitted to this hospital with a history of a right nephrectomy for a tuberculous kidney. There was a draining tuberculous sinus at the site of this previous nephrectomy. Because of the extensive bilateral advanced pulmonary tuberculosis (Fig. 3) she was placed on conservative therapy. Patient had a rapid downhill course. A bedside roentgenogram (Fig. 4) of the chest taken two days before death revealed the presence of a spontaneous pneumothorax on the right side pointing to the presence of a bronchopleural fistula.

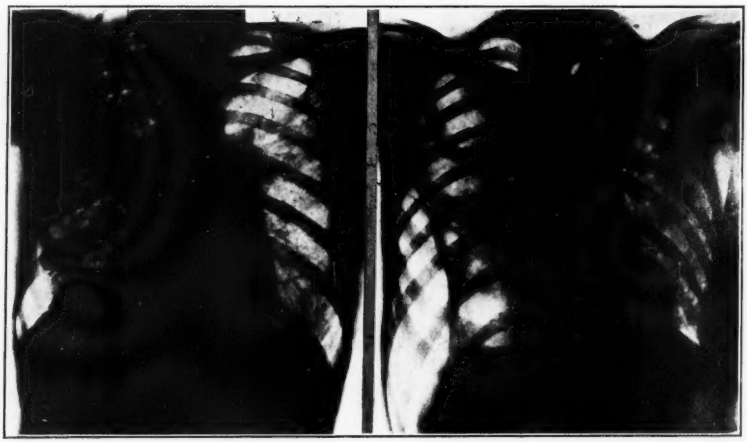

Fig. 3. Fig. 4.

Fig. 3.—Case 1. Roentgenogram demonstrating an extensive caseous pneumonic process involving the major portion of the right lung. There is also some involvement in the apex and central portion of the left lung.

Fig. 4.—Case 1. Roentgenogram taken two days before death demonstrating a spontaneous pneumothorax on the right. There is also now present an extensive caseous pneumonic lesion in the left lung with multiple excavations.

At autopsy a fistulous opening was present. This measured 1 cm. in diameter and was situated on the anterior medial border 9 cm. from the apex of the right upper lobe. This fistula communicated directly with a 2 cm. tuberculous excavation in the right upper lobe.

Comment.—This is an example of a spontaneous pneumothorax resulting from a pleural perforation in a fulminating caseous pneumonic disease process.

2. Bronchopleural Fistulas Developing in Artificially Collapsed Lungs

The great majority of cases of fistula coming to autopsy are associated with artificially collapsed lungs. The perforations in these cases

may vary in size from 1 mm. to as large as 3 cm. The patients in this group survive the fistulas for varying periods of time, therefore, in this group the opportunity is present to study the pathologic changes of the older and more chronic fistulas.

There was a total of fifty-six cases (72 per cent) in which bronchopleural fistulas formed in the course of artificial pneumothorax therapy. In order to facilitate the presentation of data in this large group an arbitrary division into two groups was made as follows: (a) those cases in which pneumothorax was the only operative procedure and (b) those cases in which thoracoplasty and Jacobaeus operations had also been performed. These latter operations were incidental procedures in that they had no pathogenetic relationship to the formation of the bronchopleural fistulas.

a. Bronchopleural Fistula Occurring in Cases in Which Artificial Pneumothorax Was the Only Operative Procedure.—There were thirty-three cases in this group, the youngest patient being 8 years old, the oldest 53, with the large majority in the 20- to 40-year age group (Table II).

Clinical diagnoses of the presence of the bronchopleural fistulas were recorded in twenty-one instances. Analysis of these cases revealed that in the great majority of instances the formation of positive pressures requiring continuous or sporadic deflations, sudden rise in temperature, increase in fluid with the formation of a mixed infection empyema, led to the diagnosis. Methylene blue was injected into the pneumothorax space in thirteen cases in an attempt to prove the presence of a communication between the pleural space and the bronchial tree. In only five cases was the methylene blue expectorated despite the fact that open fistulous communications were present at autopsy. The diagnostic criteria for bronchopleural fistulas and the efficacy of methylene blue as a diagnostic measure will be discussed in detail later.

Analysis of the duration of illness revealed a sharp demarcation into two distinct groups in that in seventeen cases the duration of illness was less than one year whereas in the remaining sixteen cases it was from one to six years with the greatest frequency in the two- and three-year groups. A review of the underlying lung pathology in these cases demonstrated that in those cases of short duration of illness (less than one year), the process was that of a fulminating caseous pneumonic tuberculosis. In those cases of long duration (one to six years) the general picture was that of chronic ulcerative pulmonary tuberculosis.

b. Bronchopleural Fistulas Occurring in Cases of Artificial Pneumothorax in Which Thoracoplasty and Jacobaeus Operations Had Also Been Performed.—There were twenty-three cases in this group. The youngest was 16 years of age, the oldest 48, with the great majority of cases in the 20- to 40-year age group (Table III).

TABLE I

DATA OF AUTOPSY CASES OF BRONCHOPLEURAL FISTULAS OCCURRING IN THE PRESENCE OF ARTIFIC

Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Age	26	41	19	24	36	35	28	53	51	20	24	20	22	37	48	35	30
Sex	M	M	M	F	M	M	M	F	M	F	F	F	M	M	F	M	M
Color	W	W	W	W	W	N	W	W	W	N	W	N	W	N	N	N	N
Duration of illness (mo.)	12	60	36	36	24	12	12	36	6	12	24	6	12	12	24	12	48
Clinical diagnosis	Y	N	Y	N	Y	Y	N	N	N	Y	Y	Y	Y	N	Y	Y	Y
Methylene blue	*NE	NI	E	NE	NE	NI	NI	NI	NI	NI	NE	NI	NE	NI	E	NI	NI
Size of fistulas (mm.)	?	L	6	5	3	30	10	3	1	6	7	10	?	10	L	0	3
Location of fistulas	LU	LU	LU	LL	LL	RU	LU	LU	LL	RU	RU	LU	LU	RU	R	U	LL
Number of fistulas	1	1	1	1	3	1	4	1	1	1	1	3	1	1	1	1	1

*For abbreviations see footnote, Table I.

Jacobaeus operations were performed in eleven cases. Three cases had no adhesions severed; one case had an apical adhesion cut and the fistulas formed in the lower lobe; and the remaining seven cases developed fistulas from varying periods of six to twelve months following the operation.

There were nine patients who had thoracoplasties performed. In four of these thoracoplasty was done as a therapeutic measure for the fistulas, and in the remaining five cases fistulas either developed after one or more stages of the thoracoplasty or the presence of the fistula was not diagnosed before the operation. In three cases both Jacobaeus operation and thoracoplasty were performed.

Clinical diagnoses of the presence of the fistulas were recorded in eighteen instances (78 per cent). Methylene blue was instilled in twelve instances and expectorated in ten of these cases. Duration of illness varied from one month to eight years, the average being from two to three years. The underlying pathology in these was that of a chronic ulcerative pulmonary tuberculosis.

Pathology and Pathogenesis.—Two types of perforations are observed, a large fistulous opening in direct communication with a tuberculous cavity and a small opening in the pleura which communicates either with a bronchus or a cavity by means of a tract.

The pathogenesis of the two types appears different. The large fistulous opening communicating with a large tuberculous cavity appears to be the result of a direct rupture of the tuberculous excavation through the visceral pleura. The appearance of the excavation in these cases is quite characteristic in that the inner surface of the cavity is formed by an irregular brown granular membrane, and trabeculae, usually seen in tuberculous cavities, are often absent. Very often the entire wall of the cavity nearest the visceral pleura is absent and the cavity opens directly into the pleural space. The perforations present in these cases usually measure between 1 and 3 cm. Occasionally, however, a small opening in the visceral pleura communicates directly with a large cavity in the lung.

TABLE I

CASES OF ARTIFICIAL PNEUMOTHORAX IN WHICH NO OTHER SURGICAL PROCEDURES HAD BEEN PERFORMED

15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33
48	35	30	15	52	37	37	32	8	38	18	19	24	21	48	47	19	20	37
F	M	M	F	M	M	M	M	F	M	F	F	M	M	M	M	F	M	M
N	S	N	N	W	W	W	N	N	N	N	W	W	N	W	W	N	W	N
24	12	48	4	72	12	36	12	12	3	6	10	48	6	36	48	8	24	12
Y	N	Y	N	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	Y	N	N
E	NI	NI	NI	E	NI	NI	NI	NI	NI	NI	NI	NE	NI	NI	E	E	NE	NE
L	0	3	1	20	†	S	†	3	20	S	10	5	30	†	†	†	20	30
R	LU	LL	LU	RU	RU	LU	RU	LU	RU	LU	RM	LU	LU	RU	RU	RL	RU	LL
1	1	1	1	2	2	1	1	1	1	1	1	1	3	1	1	3	1	2

The pathogenesis of the perforation of the larger chronic cavity through the pleura is difficult to explain. In such cavities which lie beneath the pleura there is usually a thick zone of fibrous tissue which forms the outer wall of the cavity wall and fuses with the thickened pleura. Within this wall one often sees caseous foci and it appears plausible that these caseous areas may spread to invade the rest of the cavity wall in the region of the visceral pleura, and with liquefaction the ulcerative process connects the cavity lumen and the pleural space.

The small perforation, on the other hand, appears to be the result of the liquefaction of a small caseous focus present either between the cavity wall and the visceral pleura or between a bronchus and the visceral pleura. As a result of this process there is a rupture into the visceral pleura on the one side and into the cavity or the bronchus on the other, forming a tuberculous tract (Fig. 5). These sinus tracts are narrow and measure from 1 to 4 mm. in diameter. The length of the tract may vary from a few millimeters to 1 cm.

Both the visceral and parietal pleurae are extensively thickened, the parietal membrane sometimes reaching the thickness of 1 cm. The visceral surface of the parietal pleura and the parietal surface of the visceral pleura are covered by a dense shaggy fibrin membrane. Adhesion bands so frequently observed in uncomplicated cases of artificial pneumothorax are rarely observed in cases complicated by the presence of these bronchopleural fistulas. The amount of fluid found in the pleural space at autopsy is variable, ranging from a few hundred cubic centimeters to a number of liters. The fluid usually is thick, green, and contains much fibrin. Micro-organisms are easily demonstrated and are usually the tubercle bacilli, streptococci, and staphlococci. Mixed infection empyema is a very frequent finding of bronchopleural fistulas which are present for any length of time.

The lung on the side of the bronchopleural fistula is very markedly compressed, frequently to one-third of its original size with the compression more marked in the upper lobes. The lung parenchyma is to a great

TABLE III

DATA OF AUTOPSY CASES OF BRONCHOPLEURAL FISTULAS OCCURRING IN THE PRESENCE OF ARTIFICIAL PNEUMOTHORAX AND IN WHICH PNEUMONOLYSES AND THORACOPLASTY OPERATIONS HAD ALSO BEEN PERFORMED

Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Age	34	16	32	21	29	22	39	24	21	27	30	42	26	35	32	48	38	22	24	24	23	46	37
Sex	M	F	F	F	M	F	M	M	F	M	F	M	F	M	M	M	M	F	M	F	M	M	M
Color	W	N	W	W	N	W	W	N	W	N	W	W	W	N	N	W	N	W	W	W	W	W	W
Duration of illness (mo.)	30	18	24	36	60	18	24	30	48	24	36	24	36	36	24	36	12	24	60	96	36	24	30
Clinical diagnosis	Y	N	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	N	Y	Y	Y	Y	Y	Y
Methylene blue	*E	NI	NI	NI	NI	NI	NI	NE	E	NE	E	E	E	NI	NI	NI	NI	E	E	E	E	NI	E
Size of fistulas (mm.)	4	20	7	5	20	2	?	?	3	20	40	4	15	2	25	15	10	?	L	3	?	?	?
Location of fistulas	RU	RL	LU	LU	RU	RM	LU	LL	LU	RU	LU	RU	RU	RU	LL	LL	RU	LL	RU	RU	LL	RU	RU
Number of fistulas	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1
Operative procedure	J	J	J	J	J	J	J	J	J	J	J	T	T	T	T	T	T	T	T	T	JT	JT	JT

*See footnote, Table I.

extent replaced by fibrous tissue, more particularly in the upper lobe, since the pathology is more extensive in the upper lobes. Cavities present in the lung are usually markedly compressed.

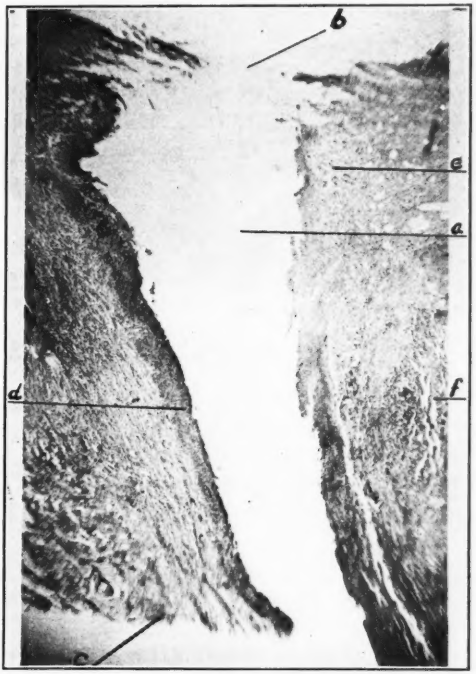

Fig. 5.—Photomicrograph of a sinus tract (*a*) between the pleural space (*b*) and the cavity wall (*c*). The inner wall of the tract is lined by a pyogenic membrane (*d*) beyond which is a vascular granulation tissue (*e*). The lung tissue beyond the cavity wall is compressed (*f*).

REPRESENTATIVE CASE REPORTS

CASE 2.—M. D., a 47-year-old white male, received a diagnosis of pulmonary tuberculosis four years before final admission to this hospital. Pneumothorax was instituted on the right side, and two years later the patient was discharged as an arrested case with negative sputum and a right hydropneumothorax. The patient worked as a kitchen man receiving refills at regular intervals for the next two years. Because of a persistent cough and loss of weight, he was examined; x-ray and physical signs revealed a spread of the disease in the contralateral lung (Fig. 6). Methylene blue was injected into the right pleural space and immediately expectorated. Bacteriologic examination of the chest fluid revealed the presence of a mixed infection. A thoracotomy was immediately performed, but the patient went rapidly downhill and died.

At autopsy a perforation measuring 2.5 cm. by 3 mm. was present 16 cm. from the apex of the right upper lobe (Fig. 7). This opening communicated directly with a system of interlocking cavities in the right upper lobe. The visceral pleura was thickened to 2 mm. and was covered with a thick fibrinous deposition.

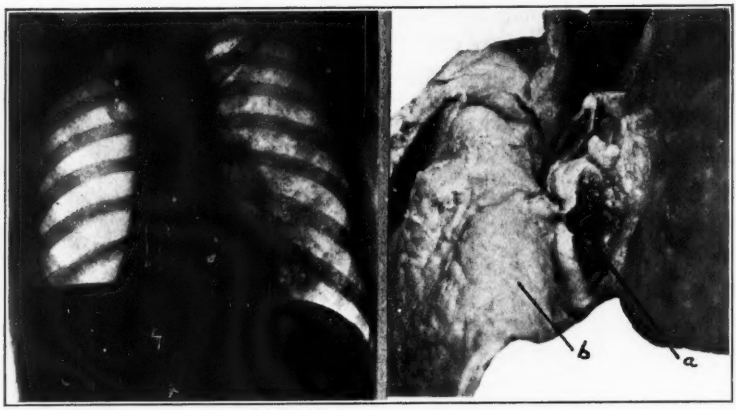

Fig. 6. Fig. 7.

Fig. 6.—Case 2. Roentgenogram taken upon readmission to hospital, two months before death. A bronchopleural fistula is present in the right lung at this time. Note the marked collapse of the right lung with the presence of a pyopneumothorax and a recent spread into the left lung.

Fig. 7.—Case 2. Gross specimen demonstrating the large bronchopleural fistula (*a*) in the right upper lobe. Note the thickened parietal pleura (*b*) covered with a dense fibrinous exudate.

Comment.—Here is an example of the formation of a bronchopleural fistula after four years of pneumothorax treatment. Although this patient was being treated as having apparently arrested case of tuberculosis, actually the process was slowly progressive, and despite the pneumothorax therapy, a large fistula formed and caused his death.

Fig. 8. Fig. 9.

Fig. 8.—Case 3. Admission roentgenogram demonstrating an extensive fulminating caseous pneumonic process occupying almost the entire left lung.

Fig. 9.—Case 3. Roentgenogram taken eight days after the institution of a left pneumothorax. Note the marked collapse of the left lung and a recent spread to the right lung. A bronchopleural fistula is present at this time in the left lung.

CASE 3.—A. C., a 29-year-old negro male, received a diagnosis of pulmonary tuberculosis and was hospitalized. Despite the fact that the patient was running a high temperature, was very toxic, and showed evidence of a caseous pneumonic process involving practically the entire left lung, a pneumothorax was advised (Fig. 8). Five days following institution of a pneumothorax, the left lung was found to be markedly collapsed, and a fluid level was present up to the angle of the scapula, and a spread of the disease was present in the right lung (Fig. 9). Patient went rapidly downhill and died three months later.

At autopsy, numerous perforations were present in the anterior medial surface of the left upper lobe. These perforations varied in size from 1.5 cm. to 3 cm. in diameter and communicated directly with a system of interlocking cavities in the left upper lobe.

Comment.—This case is an excellent example of the rapidity with which bronchopleural fistulas may develop when pneumothorax is instituted in cases of acute fulminating caseous pneumonic disease. Although the prognosis of such a case is very poor, nevertheless the formation of numerous fistulas following pneumothorax therapy makes the prognosis hopeless.

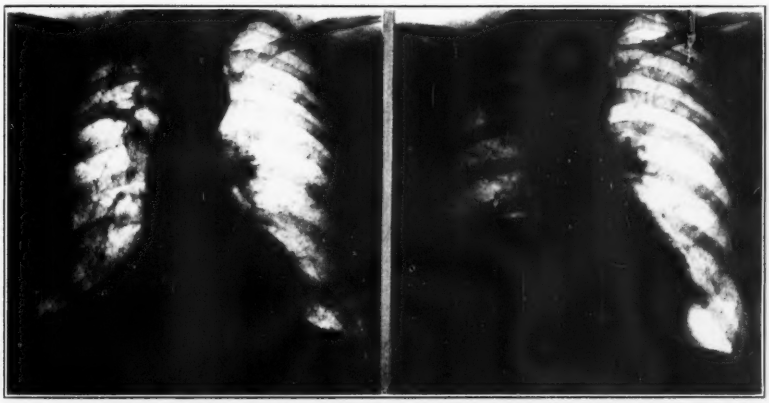

Fig. 10. Fig. 11.

Fig. 10.—Case 4. Admission roentgenogram demonstrating a caseous pneumonic process with cavitation in the right upper lobe.

Fig. 11.—Case 4. Roentgenogram taken after two years of ineffectual pneumothorax therapy. There is an encapsulated right pyopneumothorax and the right upper lobe is diffusely adherent to the chest wall. A large bronchopleural fistula in the apex of the right pleural space is present at this time.

CASE 4.—W. R., a 52-year-old white male, was diagnosed as having pulmonary tuberculosis for a period of four years received conservative treatment (Fig. 10). Sputum was always positive for tubercle bacilli, and pneumothorax was finally initiated on the right, which resulted in an encapsulated hydropneumothorax with the right upper lobe adherent to the chest wall. Intrapleural pressures were always on the positive side. The patient refused to abandon pneumothorax and have a thoracoplasty instead. Two years after the institution of pneumothorax, methylene blue was instilled into the pleural space during an aspiration of the chest fluid (Fig. 11). The dye was immediately expectorated leading to the diagnosis of a fistula. Bacteri-

ologic report of the chest fluid reported the presence of a tuberculous empyema. A thoracotomy was performed, but patient went rapidly downhill and died one month later.

At autopsy a large fistulous opening measuring 2 cm. in diameter was present in the right upper lobe which communicated with a series of large chronic cavities (Figs. 12 and 13).

Fig. 12.

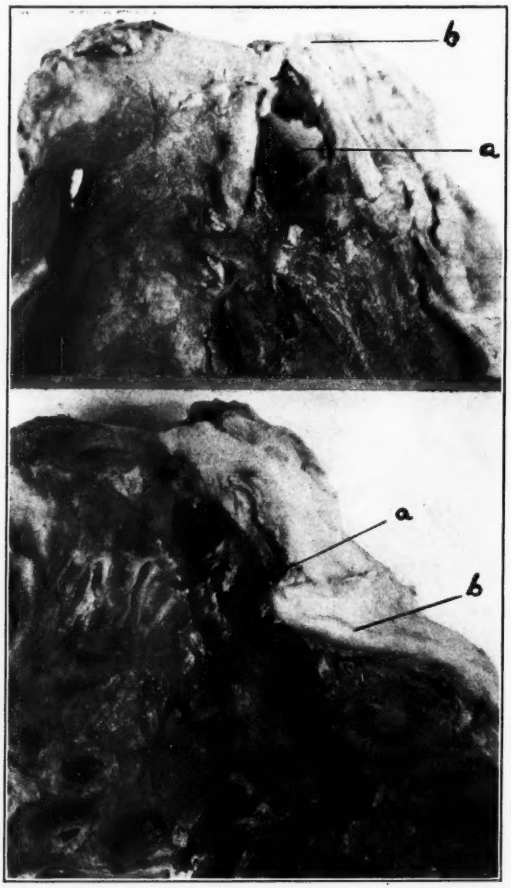

Fig. 13.

Fig. 12.—Case 4. Gross specimen demonstrating the large perforation (*a*) in the right upper lobe. Note the thickened adherent pleura (*b*).

Fig. 13.—Case 4. A sagittal section of the right upper lobe demonstrating a system of chronic cavities (*a*) which communicates with the fistulous opening in the pleura. Note the thickened adherent pleura (*b*).

Comment.—This case illustrated the danger of bronchopleural fistula present in prolonged ineffectual pneumothorax therapy.

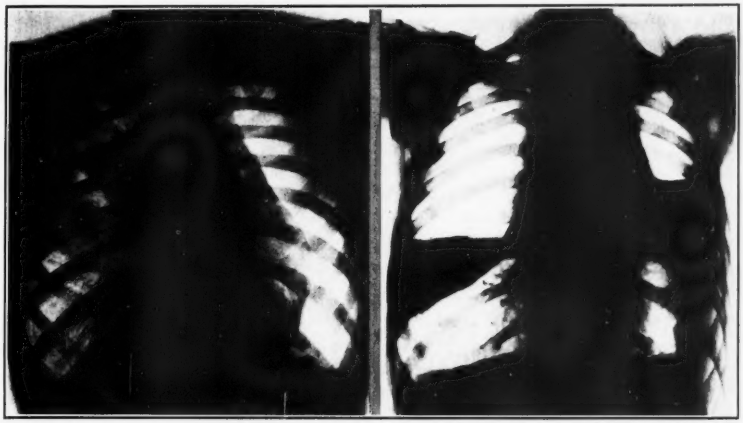

Fig. 14. Fig. 15.

Fig. 14.—Case 5. Roentgenogram demonstrating a bilateral pneumothorax with bilateral cavitation present in the upper lobe.

Fig. 15.—Case 5. Roentgenogram taken three weeks before death. Bilateral pyopneumothoraces are present with marked collapse of both lungs. A bronchopleural fistula is present on the right side at this time.

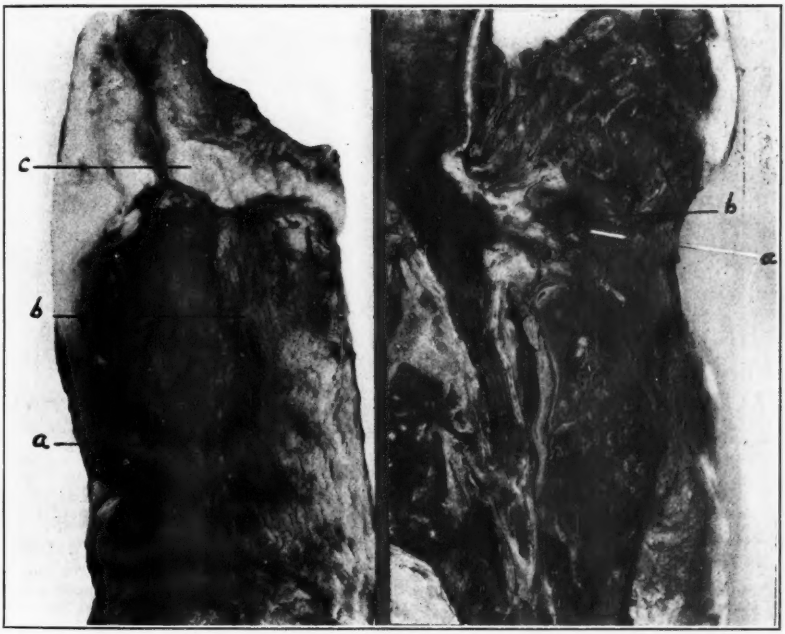

Fig. 16. Fig. 17.

Fig. 16.—Case 5. Gross specimen demonstrating a 4 mm. fistulous opening (*a*) in the right upper lobe. The visceral pleura (*b*) is thickened and covered by a shaggy membrane. A portion of the markedly thickened apical pleura (*c*) is also present.

Fig. 17.—Case 5. A sagittal section of the same case. A probe (*a*) extends from the fistulous opening into a chronic cavity (*b*).

CASE 5.—G. L., a 39-year-old white male, received a diagnosis of pulmonary tuberculosis two and one-half months before admission. A left pneumothorax was instituted which was ineffectual due to the presence of a large number of apical adhesions. A Jacobaeus operation was done and six adhesion bands were severed. Three months later a right pneumothorax was instituted which was also ineffectual because of adhesion bands. The Jacobaeus operation was then performed on the right side with the severance of four adhesion bands (Fig. 14). Empyema formed first in the left chest and later in the right. Intrapleural pressures were always negative except on the right side when positive pressures developed shortly before death (Fig. 15). Methylene blue was instilled on different occasions in both pleural spaces and was expectorated from the right side. The patient died one month later.

At autopsy both the left and right visceral and parietal pleurae were thickened and covered with fibrin. In the lateral surface of the right visceral pleura 5 cm. from the apex was a 4 mm. perforation (Fig. 16). This led into a small fistulous tract which in turn led into a tuberculous excavation in the right upper lobe (Fig. 17). No remnants of any of the adhesion bands severed previously by the Jacobaeus operation could be found.

Comment.—Clinically, this case is very interesting in that this patient had bilateral pneumothorax therapy and bilateral pneumonolyses in which a total of ten adhesion bands were severed. The right bronchopleural fistula developed one year after the pneumonolyses and there was no pathogenic relationship between the two. Pathologically, this case demonstrates the sinus tract type of bronchopleural fistulas.

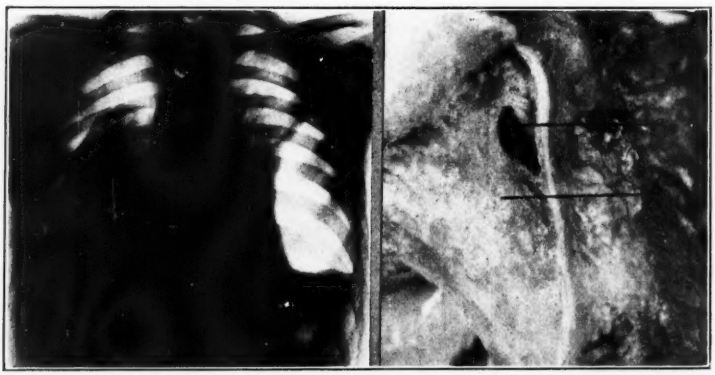

Fig. 18. Fig. 19.

Fig. 18.—Case 6. Roentgenogram taken nine days before death demonstrating encapsulated hydropneumothorax on the left limited to the lower two-thirds of the chest. The left upper lobe is broadly adherent to the chest wall and contains a giant excavation. A hydropneumothorax on the right side is also present. A bronchopleural fistula is present at this time at the apex of the left pleural space.

Fig. 19.—Case 6. Gross specimen demonstrating the large bronchopleural fistula (*a*) communicating with the large cavity in the left upper lobe. The thickened visceral pleura (*b*) has a shaggy appearance and is adherent to the parietal pleura beyond the perforation.

CASE 6.—G. B., a 25-year-old negro male, was diagnosed as having pulmonary tuberculosis six months before admission. A left pneumothorax was instituted which was ineffectual because of adhesion bands. In an attempt to control the disease in the right lung a pneumothorax was also instituted on that side. Thoracoscopy was

performed on the left, but because the entire apex was adherent to the dome of the cavity no further surgery was done. A tuberculous empyema developed on that side. An exploratory thoracoscopy was then done on the right side in an effort to obtain an effective selective collapse of the right upper lobe, and one large apical adhesion was partially severed. Intrapleural pressures were always negative until shortly before death, when positive pressures developed in the left pleural space (Fig. 18). Because of these positive pressures and extreme dyspnea the diagnosis of bronchopleural fistula was made. Methylene blue was instilled on a number of different occasions but was never expectorated. The patient had a rapid downhill course and died.

At autopsy 1 cm. from the left apical pleura on the posterior surface, was a large clean-walled opening measuring 2 cm. by 1 cm., which communicated with a large excavation in the left upper lobe (Fig. 19).

Comment.—Although a large rather chronic fistulous excavation was present at autopsy in the left upper lobe, methylene blue, instilled on a number of occasions shortly before death, was never expectorated. Pathologically this case is a good example of the direct opening of a tuberculous cavity into the pleural space.

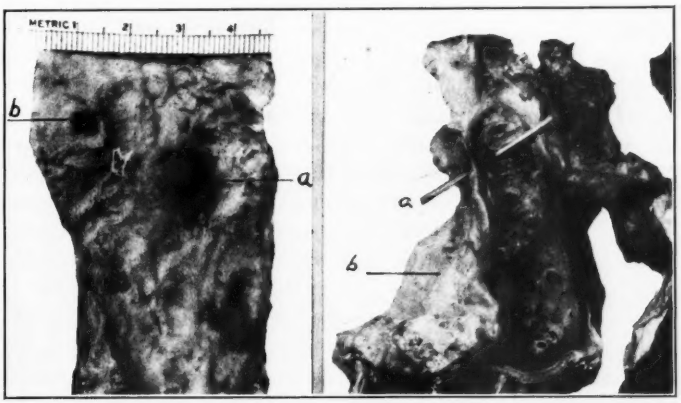

Fig. 20. Fig. 21.

Fig. 20.—Case 7. Gross specimen demonstrating a large bronchopleural fistula (*a*). Note the cross section of a large adhesion band (*b*) containing pulmonary tissue.

Fig. 21.—Case 7. Sagittal section of the same case. A probe (*a*) extends from the fistula into a cavity in the right upper lobe. The right lung is markedly compressed and the visceral pleura (*b*) is thickened and covered by a shaggy membrane.

CASE 7.—L. S., a 26-year-old white female, received a diagnosis of pulmonary tuberculosis, and right pneumothorax was instituted. After eighteen months of treatment, the patient was discharged from hospital with a tuberculous empyema as an apparently arrested case. Pneumothorax refills were continued regularly for another eighteen months, when the patient began to lose weight, coughed, and expectorated a greenish fluid. Methylene blue was instilled and immediately expectorated leading to a diagnosis of a bronchopleural fistula. A first stage thoracoplasty was done. Five days postoperatively the patient suddenly became very dyspneic and cyanotic. The right and left main bronchi were found to be occluded with thick mucoid material. The patient died before this material could be aspirated.

At autopsy a fistula was present measuring 1.5 cm. in diameter. This was located 4.5 cm. from the apex of the lung and communicated directly with a tuberculous excavation in the right upper lobe (Figs. 20 and 21). A large adhesion band measuring 8 mm. in thickness and 2.5 cm. in length was present between the right upper lobe and lateral chest wall. On section this band contained greyish-black pulmonary tissue to within 2 cm. of the parietal pleura.

Comment.—This case is another example of the progression of the tuberculous process in an apparently arrested pneumothorax case, leading to the formation of a bronchopleural fistula. This case also proves the possibility that lung tissue can be present in thick adhesion bands for a good distance of the length.

3. Bronchopleural Fistula as a Result of Closed Pneumonolyses (Jacobaeus) Operations

This group constitutes an important subdivision because of the importance and frequency of pneumonolyses as an adjunct to collapse therapy.

The formation of a bronchopleural fistula following Jacobaeus operations is the most serious complication of this procedure and causes a high morbidity and mortality. Fortunately it is a relatively infrequent occurrence. Forsee[2] collected 2,043 cases of intrapleural pneumonolyses and found that thirty-seven (1.8 per cent) developed perforation of the lung as a direct result of the operative procedure. In 650 cases of intrapleural pneumonolyses at Sea View Hospital 1.5 per cent developed perforation as a direct result of the operation.

In order to be certain that the fistula is the direct result of the operative procedure, certain requirements must be met. Clinically, symptoms of fistulas (evidenced by empyema, positive pressures, expectoration of dye, and rise in temperature) must occur soon after the operative procedure. Also there must be no suspicious evidence of its presence before operation. Pathologically, the perforation must be present in the area of the cut adhesion band. All the cases in this series met these requirements, with clinical evidence of fistula formation appearing from twelve days to one month following operation.

There were seven cases in this group, the youngest being 11 years of age, the oldest 42, with the remaining five cases between the ages of 19 and 29 years (Table IV).

TABLE IV

DATA OF AUTOPSY CASES OF BRONCHOPLEURAL FISTULAS OCCURRING AS A RESULT OF PNEUMONOLYSIS OPERATION

Case	1	2	3	4	5	6	7
Age	25	25	28	42	26	19	11
Sex	M	M	M	M	F	M	F
Color	N	N	W	N	W	N	N
Duration of illness (mo.)	36	12	96	12	18	10	24
Clinical diagnosis	Y	Y	Y	Y	Y	Y	Y
Methylene blue	*E	E	NI	E	E	NE	E
Size of fistulas (mm.)	3	30	3	40	15	10	10
Location of fistulas	RU	LU	LU	RL	RU	LU	RU
Number of fistulas	1	1	1	1	1	1	1

*See footnote, Table I.

A clinical diagnosis was made in every instance. Methylene blue was injected in six instances and expectorated in five cases. The duration of illness varied from ten months to eight years, the average being from two to three years.

Pathology and Pathogenesis.—The size of the perforations in these cases varied between 3 mm. and 4 cm. The size of these openings depends upon the size of the cavity since the excavation usually opens completely into the pleural space.

The explanation for the relatively rare occurrence of fistulas following a Jacobaeus operation lies in the structure of the adhesion bands. Adhesion bands may be divided into three distinct types: a thin cordlike band composed entirely of hyalinized connective tissue, a broader round band containing pulmonary tissue for varying extents toward the parietal attachment, and fanlike adhesions always containing lung tissue to its parietal attachment. The fanlike adhesions are dangerous to sever and are usually left intact by the surgeon. The great majority of bands are the thin hyaline connective tissue bands devoid of lung tissue. Section of these bands at their parietal attachment leads to a necrosis of the band which halts at its visceral junction. Proof of this is found at autopsy since in a great number of cases, all traces of the bands severed by previous Jacobaeus operation are absent. The round adhesion bands containing lung tissue are the ones that probably cause fistula formation, since either the coagulative necrosis extends to the pulmonary tissue or the lung tissue is cut directly leading to a bronchopleural fistula. Since these adhesions are usually found over the site of the cavities, the necrosis continues to invade the lung tissue until the cavity becomes involved and this in turn opens directly into the pleural space.

REPRESENTATIVE CASE REPORTS

CASE 8.—T. J., a 42-year-old negro male, was diagnosed as having pulmonary tuberculosis, and a right pneumothorax was initiated. A Jacobaeus operation was performed on this side in an attempt to sever apical adhesions preventing collapse of the cavity in the right upper lobe (Fig. 22). Thoracoscopy report was as follows: "In the ordinary position only one adhesion was visualized. This was severed and patient was turned on his left side where a second adhesion was visualized. This adhesion extended far back posteriorly and the lung was seen to be adherent to the chest wall posteriorly. This adhesion was separated to a large part of its extent but complete severance was impossible." Following operative procedure the temperature was spiking in character up to 103° and three weeks following operation positive pressures necessitating deflation of air were present. Methylene blue was instilled and immediately expectorated. Bacteriologic examination of the empyema revealed the presence of a mixed infection. The patient had a rapid downhill course and died one month later.

At autopsy a large fistulous opening was present in the right upper lobe communicating directly with a chronic cavity (Fig. 23).

Comment.—In this instance a bronchopleural fistula formed approximately three weeks following the Jacobaeus procedure. The site of the perforation corresponded with the location of the adhesion which was only partially severed previously.

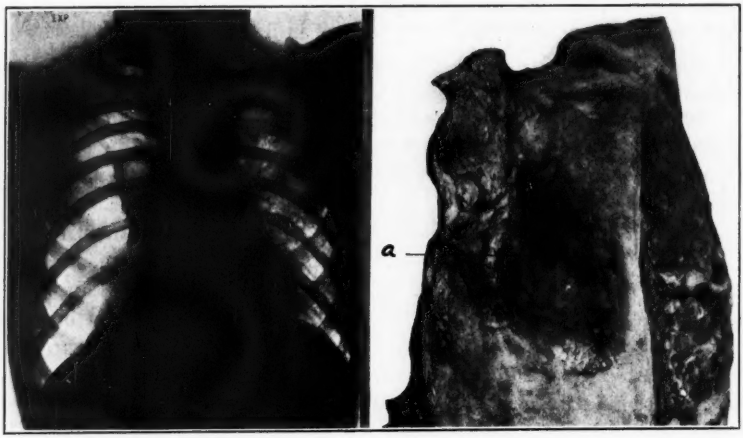

Fig. 22. Fig. 23.

Fig. 22.—Case 8. Roentgenogram demonstrating irregular collapse of the right lung. Note the large apical cavity held open by numerous adhesion bands to the dome of the right pleural space.

Fig. 23.—Case 8. Gross specimen demonstrating a 4 cm. fistulous opening (*a*) communicating directly with a large cavity. This fistula is the direct result of a Jacobaeus operation.

CASE 9.—J. G., a 28-year-old white male, received a diagnosis of pulmonary tuberculosis and was placed on conservative therapy. The sputum turned negative and remained so for a period of seven years. Because of an exacerbation of the disease and presence of a positive sputum, a pneumothorax was initiated on the right. This was ineffectual due to broad apical adhesions. A contralateral pneumothorax was started on the left in an attempt to control the disease in that lung. A Jacobaeus operation was performed on the right side and the thoracoscopy report was as follows: "There is a broad wedge shaped adhesion attaching the apex to the dome of the chest wall postero-laterally. This wedge shaped adhesion, triangular in outline, has a base formed by the mediastinum and extends toward the dome of the chest wall and attaches itself to the first rib. The reinforcing bands make this adhesion broad at either end and somewhat narrowed in the mid portion. About two inches of this adhesion was dissected free from the chest wall by means of splitting the parietal pleura and carefully decollating with the cold cautery." Following the operative procedure fluid developed and there was a sharp rise in temperature to 101°. Two weeks later positive pressures began to develop which necessitated continuous deflation. Pneumothorax was continued on the left until one month before death, when a traumatic spontaneous pneumothorax developed in that side necessitating simultaneous deflation of both pleural spaces. The patient went progressively downhill and died. The bacteriologic report of the right empyema revealed the presence of a mixed infection.

At autopsy a number of firm adhesion bands were present in the lateral aspect of the right pleural space. One of these was situated 3 cm. from the apex and measured 1.5 cm. in diameter. Another large adhesion band just basal to this one measured 3 cm. in length and was fused with the interlobular fissure. At the junction of this band with the visceral pleura there was a perforation into the right upper lobe measuring 3 mm. in diameter. A traumatic fistula in the left lung was not discernible at autopsy.

Comment.—This type of adhesion band (broad, fanlike) is usually not suitable for pneumonolysis, and in this case a perforation developed as a result of the operative procedure.

DISCUSSION

The Relationship Between the Duration of Pneumothorax Treatment and Formation of Bronchopleural Fistulas.—In reviewing the clinical and pathologic findings in this series it soon became evident that bronchopleural fistulas complicating artificial pneumothorax therapy fitted into two main divisions. In one group fistulas developed very shortly (six months or less) after the initiation of pneumothorax, as contrasted with the other group in which fistulas developed in from one year to four years of pneumothorax therapy (Fig. 24).

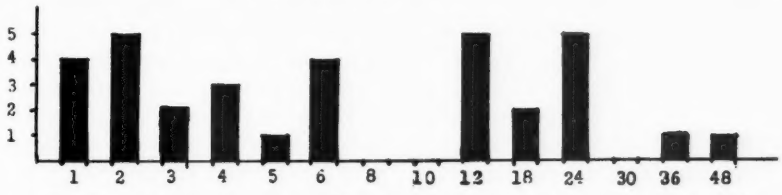

Fig. 24.—Analysis of cases according to the interval of time from the initiation of pneumothorax to the diagnosis of bronchopleural fistulas. Numbers at the left indicate number of cases; numbers at bottom, time period in months.

There was a total of thirty-three cases in which a definite correlation between the time of formation of the fistula and length of pneumothorax therapy could be made. In nineteen instances, fistulas developed in less than six months after the initiation of pneumothorax, with nine cases developing fistulas in less than two months. In the remaining fourteen cases fistulas developed in from one to four years of pneumothorax treatment.

The pathologic processes in the lungs of those cases in which fistula developed very shortly after the initiation of pneumothorax consisted of an acute fulminating caseous pneumonic tuberculosis. In the cases which developed fistulas after one to four years of pneumothorax therapy, the pathologic picture was that of chronic pulmonary tuberculosis. There have been in the main two schools of thought concerning the initiation of pneumothorax in the acute exudative pneumonic tuberculous processes. One group believes that pneumothorax should not be attempted until liquefaction and sloughing of the caseation and fall in temperature have occurred. Ornstein and Ulmar[3] stated that it is useless to attempt pneumothorax at an acute early stage as not only will sloughing take place as in the uncollapsed lung, but this sloughing will extend to the visceral pleura with the subsequent formation of a fistula. The other group does not consider early acute exudative processes a contraindication to collapse therapy (Eglee and Jones[4]). Our findings ap-

parently corroborate the observations of the former group. Although the prognosis of cases with acute fulminating caseous pneumonic disease is poor, nevertheless, the morbidity and mortality are greatly increased with complicating bronchopleural fistulas. It is probably better, therefore, to wait for sloughing of the caseous process and for organization of the perifocal inflammation into fibrosis and subsequent thickening of the pleura to occur in such case before initiation of pneumothorax treatment.

Bendove, Miller, and Alexander[5] have demonstrated that if a pneumothorax does not effect a conversion of a positive into a negative sputum within six months of treatment the chances that it will occur after that period are approximately one to five. Generally at this hospital pneumothorax is abandoned after a six months' period, if it does not effect a conversion of the sputum, in favor of other surgical procedures such as thoracoplasty. The formation of bronchopleural fistula in cases after one to four years of pneumothorax treatment adds another strong indication for the abandonment of ineffectual pneumothoraces early and the substitution of some other surgical procedure.

The Relationship of Empyema and Bronchopleural Fistulas.—Empyemas are a constant accompaniment of bronchopleural fistulas except in those cases in which the fistulas develop so soon before death that there is not sufficient time for the empyemas to develop.

A mixed infection empyema is found most often in the presence of a bronchopleural fistula. Pyogenic organisms can be introduced into a tuberculous empyema either from outside the chest wall (by means of aspirations) or from the inside of the thorax through the bronchopleural fistulas. The former route is so uncommon, however, that it can be considered negligible, and when a mixed infection is reported we can usually postulate and prove the presence of a fistula. It is important to bear in mind that bronchopleural fistulas are also present in the absence of a mixed infection empyema.

In this series empyemas were present in every instance, with the exception of five cases in which death occurred too soon for the empyema to develop. The reports of the bacteriologic examinations performed antemortem revealed the following incidence (Table V).

The pyogenic organisms usually present were streptococci and staphylococci, with an occasional instance of pneumococci and Friedlander's bacilli being reported.

TABLE V

ANALYSIS OF BACTERIOLOGIC EXAMINATIONS OF EMPYEMAS COMPLICATING BRONCHOPLEURAL FISTULAS

BACTERIOLOGIC REPORTS	NO. OF CASES
Mixed tuberculous and pyogenic empyemas	27
Pure tuberculous empyemas	17
No aspirations performed	28
Inadequate reports	6

There are a number of important points concerning relationship of bronchopleural fistulas and empyema which should be considered. Coryllos[6] has postulated that all tuberculous empyemas are caused by the rupture of a caseous subpleural focus into the pleural space with the subsequent formation of a pleuropulmonary fistula. These may vary in size from small punctiform to large fistulas, with the great majority being the punctiform type. Since these are very small they usually give very slight clinical evidence of their presence and usually heal. This hypothesis is difficult to substantiate pathologically because at autopsy it is very difficult or impossible to prove the presence of healed fistulas. The thickened pleura with its fibrinous coating adequately obliterates all evidence of the presence of any former fistulas.

A moot question which is often raised is what is the effect of a long-standing tuberculous empyema on the visceral pleura and lung parenchyma? Can such empyemas ulcerate through the visceral pleura and produce a fistulous tract? At the present time investigative work is being done on the various aspects of the relationship of empyema to fistula formation and will be the subject of a future report.

Diagnosis of Bronchopleural Fistulas.—A clinical diagnosis of the presence of a bronchopleural fistula in the great majority of cases can be usually made providing the possibility of its existence is considered. An early diagnosis is of prime importance so that adequate treatment can be immediately instituted in order to avoid the severe complications (mixed infection empyemas, dyspnea, rapid spread of disease due to aspiration of chest fluid), which are responsible for the high mortality in these cases.

There are a number of prominent symptoms and signs which are of aid in the diagnosis. A very early and prominent symptom in a large majority of cases is a sudden and marked increase of intrapleural air leading to positive manometric readings and requiring deflation of the air. Concomitant with the increase of pleural pressure, one finds a marked increase of almost complete collapse of the lung. There is also usually a mediastinal herniation to the contralateral side with a shift of the mediastinal structures in those cases with a mobile mediastinum. If no fluid is present in the pleural space prior to the formation of the fistulous opening, it very rapidly forms and usually goes through a rapid evolution from a thin serous type to a thick pus in which tubercle bacilli can be isolated and in most instances pyogenic organisms. In those cases with hydropneumothorax, the formation of the fistula is generally evidenced by a rapid increase of fluid and the development of a tuberculous or mixed infection empyema. A symptom, which in many cases is very valuable, is the change in the character of expectorated material. A patient with a bronchopleural fistula will often volunteer or in response to questioning give the information that the expectorated material has suddenly increased in quantity and changed in consistency becoming

thinner and appearing similar to chest fluid. In many instances he will know that this phenomenon occurs only when he assumes a certain position in bed and this is a clue as to the location of the fistula in the pleural space.

A sudden sharp spiking rise in temperature with new extensive physical signs of involvement of the contralateral lung should make one immediately suspicious of fistula formation with spill to the contralateral side. An important and often early physical sign is the presence of a metallic tinkle. This auscultatory sign is usually produced by the bubbling of air from a fistula into the chest fluid. When this sign is present one should be immediately suspicious of the presence of a bronchopleural fistula. The roentgen-ray examination of the chest is important for verification of the clinical picture.

It is important to bear in mind that in many instances, diagnosis of bronchopleural fistula is very difficult and that the clinical findings and symptoms may be very indefinite. This is especially true in those cases with very small fistulas which may cause only slight modifications of pleural pressures and functional respiratory symptoms.

In this series clinical diagnosis of the presence of a bronchopleural fistula was made in fifty-two of the seventy-eight cases (67 per cent). In the review of these cases, it was evident that more cases would probably have been diagnosed if the physician in charge had considered the possibility of the presence of a fistula. Also, as described previously, in those cases of fistula formation complicating fulminating massive caseous pneumonic disease fistulas form very shortly before death and in most instances the diagnosis is not or cannot be made because of the obviously hopeless and critical condition of the patient.

At the present time the instillation of a 1 per cent aqueous solution of methylene blue into the chest cavity is the most common procedure used for verification of a clinical diagnosis. If the dye is expectorated relatively soon after its injection, it can be reasonably assumed that a bronchopleural fistula is present. On the other hand, if the dye is not expectorated, one cannot be assured that a fistula is not present. In this series methylene blue was injected in thirty-four instances of clinically suspected cases, but the dye was only expectorated in twenty-two instances (65 per cent), despite the presence of a fistula at autopsy. There was no apparent relationship between the size of fistula and the expectoration or lack of expectoration of the methylene blue. A possible explanation for the failure of the dye to appear in the sputum in certain instances is the location of the fistula at the extreme apex of the pleural space where the dye may not come in contact with the opening. Occasionally methylene blue may be reduced in the empyema fluid to a colorless leuco-base and the possibility exists that it may be present in the sputum as such. Therefore, it is advisable to add to the sputum of such suspected cases an oxidizing agent such as hydrogen peroxide

which may restore the blue color. Methylene blue will not be expectorated in cases of open fistula if there is blockage of the bronchi. In one instance in which methylene blue was instilled a number of times in a case in which the presence of a fistula was strongly suspected, no dye was ever expectorated. This patient was maneuvered into a number of positions in order to assure contact of the dye to all surfaces of the pleural space. At autopsy, a large fistulous opening was present measuring 2 cm. by 1 cm. In this case the only possible explanation for the lack of dye expectoration was a blockage of the bronchus which prevented the escape of the dye.

Quantitative analysis of the oxygen and carbon dioxide gases in the pleural space has been advocated by Coryllos[6] as a diagnostic measure for bronchopleural fistulas. The procedure is now being used routinely at this hospital in all cases in which bronchopleural fistulas are suspected. An increase in the percentage of oxygen and usually a decrease in the percentage of carbon dioxide are considered indicative of a fistulous communication between the pleural space and the bronchial tree. In this series, however, there was only one case in which gas analyses of the pleural air had been done before the patient came to autopsy. In this case, three gas analyses were done at varying intervals from two to three months before the development of the fistula. All these showed oxygen to be present in very small amounts (0.29 volumes per cent, 0.24 volumes per cent) with carbon dioxide values within normal limits (7.5 volumes per cent to 9.8 volumes per cent). Immediately after the diagnosis of fistula was made clinically (expectoration of chest fluid and methylene blue), gas analysis revealed almost 11 volumes per cent oxygen. At autopsy a large perforation measuring 2.5 cm. by 1.5 cm. was present.*

Healing of Bronchopleural Fistulas.—In certain instances, temporary closure or permanent healing of bronchopleural fistulas is known to occur.

Clinically many cases are seen with definite evidence of the presence of a bronchopleural fistula which apparently close permanently. At autopsy it is difficult to study the pathologic changes occurring in the healing of these fistulas, since the thickened pleura usually adequately obliterates all evidence of their presence. Occasional cases, however, are observed at autopsy in which certain of these changes can be studied.

Healing of a fistula occurs in one of two ways. The visceral pleura may become adherent to the parietal pleura in the region of the perforation, or the exudate covering the visceral pleura may extend to seal the opening.

Healing of the first type may be accomplished by the expansion of the lung so that the visceral and parietal pleuras become fused to entirely

*Gas analysis in this case was performed by Dr. Allen Welkind.

seal off the perforation. This type of healing is important for the closure of the larger perforations. Thoracoplasty operation for closure of bronchopleural fistula attempts to utilize this type of healing.

Healing of the second type usually occurs in the smaller fistulas. A serofibrinous exudate results from the emptying of the contents of the liquefied area into the pleural space. This exudate on the visceral pleura

Fig. 25.

Fig. 26.

Fig. 25.—A fibrinous "cap" (*a*) on the visceral pleura sealing off a small bronchopleural fistula.

Fig. 26.—The same view with the fibrinous "cap" removed demonstrating the bronchopleural fistula (*a*).

extends to cover the small perforation and is later transformed into fibrous tissue. At this stage of development it is difficult and often impossible to find the site of perforation, despite the fact that there had been definite clinical evidence of its presence. Healed perforations at this stage cannot be distinguished from subpleural cavities which cause

a thickening of the overlying pleura. In a recent autopsy case evidence of this method of healing was demonstrated clearly. The patient expired two days after a spontaneous pneumothorax and at autopsy a 2 mm. fistulous opening was hidden and sealed by a fibrinous cap. Upon removal of the fibrin an open fistula was found (Figs. 25 and 26).

Temporary closure of a fistula may be explained by the temporary presence of fibrinous plug which also may ulcerate or become detached and again leave an open fistula.

SUMMARY AND CONCLUSIONS

1. A review of 1000 consecutive autopsies of chronic ulcerative pulmonary tuberculosis revealed the presence of bronchopleural fistulas in seventy-eight cases (7.8 per cent).

2. The bronchopleural fistulas are classified into three groups as follows: (1) fistulas occurring in previously uncollapsed lungs (15 cases), (2) fistulas occurring in the presence of an artificial pneumothorax (fifty-six cases), and (3) fistulas as a direct result of closed pneumonolyses (seven cases).

3. The pathology and pathogenesis of each of these groups are discussed.

4. The size of the fistula at autopsy varied from 1 mm. to 4 cm., with the great majority (73 per cent) present in the upper lobes of the lung.

5. Clinical diagnoses of the presence of the fistulas were made in fifty-two cases (67 per cent). Methylene blue was injected in thirty-four cases and expectorated in twenty-two of these. The criteria for a clinical diagnosis are discussed in detail.

6. The relationship between the pneumothorax treatment and the formation of a bronchopleural fistula is discussed. Cases of bronchopleural fistulas occurring within six months after the induction of artificial pneumothorax usually had a fulminating caseous pneumonia. The underlying lung pathology in cases of bronchopleural fistulas occurring after one to four years of pneumothorax therapy was that of a chronic pulmonary tuberculosis.

7. Empyemas were present in every instance with the exception of five cases, in which the fistulas developed too soon before death for fluid to develop. Mixed infection empyemas were recorded in twenty-seven instances, tuberculous empyemas in seventeen, and the remaining cases either had no aspirations or the reports were inadequate.

8. Healing of bronchopleural fistulas may be temporary or permanent. Healing may occur as a result of visceral pleura becoming adherent to the parietal pleura in the region of the perforation, or a fibrinous exudate which later is organized into fibrous tissue may seal off the opening.

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DISCUSSION

DR. J. SMITH, PUERTO RICO.—I was very much impressed with the low incidence of bronchopleural fistulas occurring in cases following pneumolysis in Sea View Hospital. I know the extensive resection that is sometimes done, particularly in cases of bilateral disease where an effective pneumothorax collapse had to be obtained before attempting a collapse on the contralateral side.

In this connection I would like to mention my experience in Puerto Rico. Because of the large amount of pulmonary tuberculosis and the comparative shortage of hospital beds, it has been necessary to attempt to convert as large a number of patients into a satisfactory ambulatory status as rapidly as possible, with the result that we have attempted to do almost routine early pneumonolyses in cases of pneumothorax with adhesion. We have done 480 in the past ten months. Of the first 200 consecutive cases we have had one bronchopleural fistula develop as a result of the operative procedure.

DR. POL N. CORYLLOS,* NEW YORK CITY.—It gives me great pleasure to discuss the excellent paper of Dr. Auerbach and Dr. Lipstein. Dr. Auerbach is the Director of the pathologic laboratory of Sea View Hospital. His thorough work has been of invaluable help to the medical and more especially to the surgical staff of this great institution. This is especially true in the management of tuberculous empyemas. My work which was published last year in our journal was greatly helped by the pathologic findings of Dr. Auerbach which he has reviewed today and which will appear in detail in his published paper.

Two facts are especially to be retained: the frequency of bronchial fistulas in tuberculous empyema and the importance of the size of these fistulas in the evolution and the prognosis of the empyema. It is obvious that in large fistulas, representing ruptures of tuberculous pulmonary cavities into the pleural cavity and communication with the latter of large bronchi, massive tuberculous infection of the pleura occurs, often complicated by pyogenic superinfection. The prognosis in these cases is as a rule desperate. On the contrary, when only small bronchial fistulas are formed the prognosis is better and inversely proportionate to the size of the fistula. Small fistulas frequently close and open pneumothorax is transformed again in closed pneumothorax. Intrapleural pressure from positive becomes negative, dye injected into the pleural cavity is not expectorated, and turbid exudate may change again to clear fluid and become absorbed. These facts show the tremendous importance in the timely and accurate diagnosis of the presence and the size of bronchial fistula by gas analysis of the pleural air.

Regarding the production of bronchial fistulas as a complication of closed pneumonolysis we had less than one per cent in our series although extensive and staged

*Deceased.

operations had been carried out during the last four years, in bilateral cases. In previous years the percentage of these complications was higher. They have decreased to almost vanishing point since the new instrument which I had devised has allowed me to dissect the adhesions away from the parietal pleura by carrying the dissection in the subpleural space. Remarkable clinical results have been obtained by this simple procedure; at the same time, the number of injuries to the lung and production of bronchial fistulas have ceased almost completely.

I want to express again to Dr. Auerbach and Dr. Lipstein my congratulations for this remarkable and thorough work. Investigations of this kind are of inestimable value to the thoracic surgeon.

It gave me great pleasure to hear the remarks of Dr. Jacob Smith, acting Chief Surgeon of the Hospitals for the tuberculous patients, of Puerto Rico. Three years ago Dr. Smith was my chief Resident Surgeon at Sea View Hospital. It is a real thrill and satisfaction for me to appraise the excellent work which he is doing in the new center which he has created. It is in the tradition of our Society to welcome and encourage our young men who are destined to propagate and improve on our ideas and methods and to devote their lives to the help of tuberculous patients.

DR. DEAN B. COLE, RICHMOND, VA.—I was very much impressed by the paper of Dr. Auerbach and Dr. Lipstein. They have certainly shown us the end results of empyema.

The point I want to emphasize was brought out by them—the large number of patients dying of tuberculous empyema who were never aspirated. I feel that there should be more aspirations, fewer autopsies. For a long time we have been convinced that if these patients are treated early and adequately, many if not most severe bronchopleural fistulas are avoided.

If those of us giving pneumothorax would aspirate all fluids, certainly determine whether or not they are purulent and if they are purulent treat them promptly, frequently, and adequately, we would avoid many complications and sequelae.

DR. OSCAR AUERBACH, NEW YORK CITY.—I think that our study revealed the important fact that the majority of bronchopleural fistulas developed in cases in which artificial pneumothorax had been instituted.

There were sixty cases which we studied but which were not included in this series. These were cases of mixed infection empyema which could not be tabulated with the seventy-eight cases here reported because at the time of autopsy they did not have a bronchopleural fistula. Many of these sixty cases showed definite clinical evidence of the presence of a bronchopleural fistula during their course. It appears likely that after the emptying of the bacteria into the pleural space when the subpleural focus ruptures through the visceral pleura the perforation is sealed off. After the perforation is closed the mixed infection empyema remains. In view of the fact that the majority of these cases also occurred in the presence of artificial pneumothorax we must conclude that this type of therapy not infrequently leads to a severe complication.

LATE RESULTS OF LOBECTOMY FOR BRONCHIECTASIS IN CHILDREN

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OPINION differs regarding surgical treatment of some cases of bronchiectasis and particularly of a certain group occurring in childhood. In this paper, for the convenience of presentation, the cases are divided into three groups; first, those in an advanced stage; second, those with moderate involvement; third, those whose symptoms are negligible and a diagnosis is possible only by careful physical and bronchogram examinations.

In the two extremes no difference of opinion exists. All are agreed that surgical extirpation of the involved portion of the lung is the treatment of choice in patients who are in the advanced stage, with large bronchiectatic abscesses, who are expectorating huge quantities of malodorous sputum, and whose mental and physical development is retarded. In early cases where bronchial dilatation involves a very small portion of the lung, where a diagnosis cannot be made other than with a bronchogram, where the sputum is small in amount, where physical development is normal, all agree they should be treated conservatively with postural, supplemented by bronchoscopic, drainage. But we find conflict existing between the surgeon and the pediatrician in the middle group; where there is a moderate involvement of a lobe or lung, where sputum is profuse and somewhat purulent, where normal development still obtains, and definite improvement, even though temporary, follows conservative measures, the selection of appropriate treatment has not as yet been standardized.

Published mortality statistics, especially those from the large clinics, demonstrate conclusively that the mortality in lobectomy and pneumonectomy is, at the present time, sufficiently low so that hesitation to recommend operation on that basis is not tenable. The pediatrician hesitates not only because of the operative mortality, which he is inclined to exaggerate, but also because he fails to appreciate the effect of a successful operation on the condition of the remaining pulmonary tissue, on the general development of the child, on its effect on the vascular system, whether postural deformities occur, and whether the cure is permanent or relapse may be expected.

Read before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

From the Children's Surgical Service, Bellevue Hospital, Dr. Fenwick Beekman, Director.

With these questions still unanswered, a critical survey was made of the four cases reported before the American Association for Thoracic Surgery in 1934, as well as those subsequently operated on for lobectomy or pneumonectomy. As a group, all these patients have been seen in the follow-up clinic at least twice a year, where their general health, their development, and the amount, if any, of daily sputum were investigated with generally quite satisfactory results. However, on more careful scrutiny, complications and sequelae were found, which examination in the return clinic failed to disclose. These patients were then readmitted to the ward for careful physical, x-ray and bronchogram examinations, together with an electrocardiogram and vital capacity.

All cases operated on in the Children's Clinic at Bellevue Hospital belonged to the first described group. They were advanced cases which could not be benefited by any form of conservative treatment.

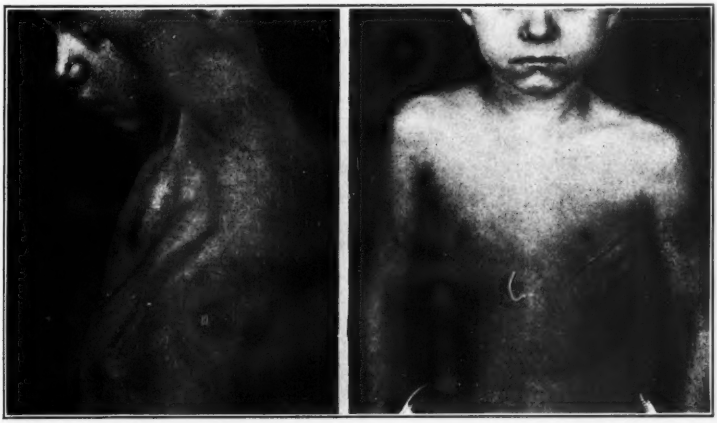

Fig. 1.—Case 1, after pneumonectomy in two stages, showing absence of deformity. Left, patient on Feb. 24, 1938, and right, March 9, 1938.

CASE 1.—H. W., a 5-year-old girl, whose entire left lung was bronchiectatic, was first operated upon in January, 1936. Through an anterolateral incision, the left chest was explored. Both the lower and upper lobes were adherent to the parietal pleura; the interlobar fissure was easily found and was almost free of adhesions. Both lobes were shrunken, fibrotic, and contained but little air. Due to the dense adhesions, it was believed wise to remove only the lower lobe, leaving the upper for a second operation.

Convalescence from this lobectomy was uneventful. The child was out of bed on the sixteenth day; no bronchial fistula developed, and the amount of cough and sputum was markedly diminished. Removal of the upper lobe was delayed on account of an attack of toxic diphtheria, with threatened myocardial disturbances, and a tonsillectomy for subsequent hypertrophy and infection of the tonsils.

In October, 1937, the chest was reopened through an anterolateral incision. No ribs were resected but the third and fourth were divided anteriorly. The upper lobe was atelectatic and fibrotic. It occupied the superior sulcus. The diaphragm was

elevated but not adherent to the upper lobe. The entire mediastinum was shifted to the left. The heart could be retracted readily to its normal position but further displacement was followed by a marked increase in the pulse rate. No other abnormality of the heart was noted.

The lobe was easily removed, using a Shenstone tourniquet threaded with copper fishing line. (This serves admirably as a tourniquet. It does not stretch, has tensile strength, and yet does not cut into the tissue when the instrument is snugly applied.) The stump was closed by ligation of the individual vessels with chromic gut and the entire mass buried by suture of the overlying parietal pleura. The space was drained, because of the possible contamination from the cut bronchi. There was serosanguineous discharge, but no empyema developed. The phrenic nerve was not interrupted. A bronchocutaneous fistula developed but closed spontaneously. The pleural space was completely obliterated by herniation of the right lung, shifting of the mediastinum, marked elevation of the left diaphragm, and narrowing of the intercostal spaces with flattening of the wall. A definite vesicular murmur can now be heard over the lower portion of this patient's chest, and a bronchogram shows the right lung well beyond the left sternal line. The patient, now 6 years of age, is in excellent health and leads a normal existence. Electrocardiogram shows a left axis deviation but no other abnormality. Vital capacity was not taken due to lack of cooperation of the patient. Bronchogram of the right lung is normal.

In an excellent article, Rienhoff[1] has shown conclusively that the contralateral lung is able, by compensatory dilatation, to help obliterate the space, as well as to compensate physiologically for the resected lung. This compensatory dilatation and simultaneous herniation of the remaining lung were apparent within one month following the complete pneumonectomy.

CASE 2.—F. A., a girl 10 years of age, was first operated on in 1933. She had an advanced saccular bronchiectasis of the left lower lobe, which, at operation, was freed from its dense adhesions. As this constituted a major procedure, it was deemed unwise to remove the lobe in a single stage.

At the second stage, three weeks later, no line of cleavage could be found, because of the unusual formation of very dense adhesions. The lobe was particularly adherent to the diaphragm and could not be safely separated. As profuse secretion made removal of the lobe imperative, a double row of clamps was placed as near the hilus as possible, and the main portion of the lobe was resected by cutting through the lung tissue. The pedicle was thus freed and mass ligated. It was believed that the small portions of adherent lung would undergo spontaneous separation after ligation of the hilus but they remained viable, drawing their blood supply from the structures to which they were adherent. Closure of the bronchus at the second stage resulted in immediate cessation of expectoration, though quantities of mucopus drained from the chest cavity. In other words, the separate pieces of lung not only remained viable, but the bronchiectatic abscesses in those portions continued to produce the same type of mucopus that had previously been expectorated.

Gradually, the operated area closed, forming a latticework with multiple fistulas leading to one cutaneous opening. Clinically, the result, other than the discharging sinus, was satisfactory. The patient became healthy, clubbing of the fingers receded, and physical and mental development progressed normally. The persistence of the fistula and of the foul abscess contents demanded a reopening of the chest and resection of the remaining fragments of lung tissue. A vessel 2 mm. in

diameter was found nourishing the portion attached to the diaphragm. Histologically, these separate specimens were atelectatic, abscessed, tuberculous lung tissue, although the main portion of the lung first removed did not show a tuberculous involvement.

This patient is now 15 years of age, has normal mental and physical development, and is clinically well, with the following record: vital capacity, 1978 c.c.; calculated vital capacity, 2300 c.c.; tidal air, 370; electrocardiogram, normal; bronchogram (of remaining pulmonary tissue), normal.

The case just cited was the sole exception to the general observation that adhesions found at the second operation are soft, easily separated, and bleed only slightly. With modern technique and anesthesia, two-stage lobectomies will become less frequent, but when they are indicated the possibility of dense adhesions forming between stages must be kept in mind. A very similar experience was reported by Beye,[2] in 1935, who presents a case in which the entire lung remained viable after resection of the hilus.

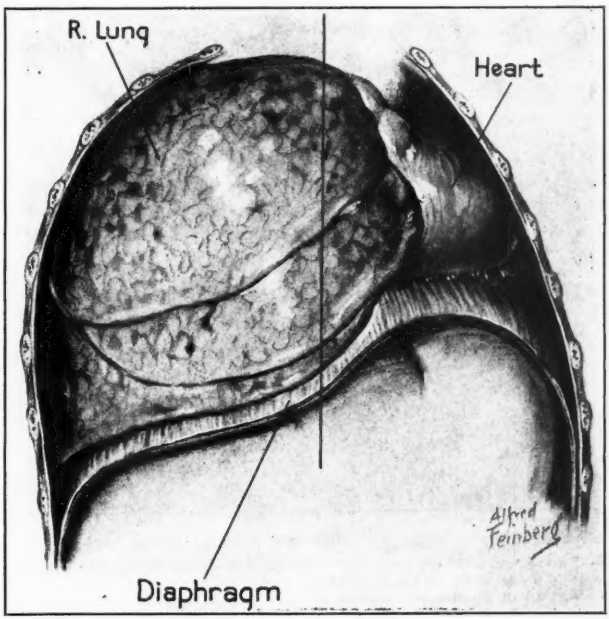

Fig. 2.—Case 3. Sketch made at autopsy showing enlargement of right lung with protrusion into left thoracic cavity, elevation of diaphragm, and shift in position of heart. Because of the shrunken and atelectatic condition of the remaining left upper lobe this closely represents the condition found at the second operation.

CASE 3.—A. B., a boy 12 years of age, at the time of his operation, 1932, had an involvement of the entire left lower lobe, with bronchiectasis. The lingula and a small portion of the body of the upper lobe were also involved. Following the lower lobe resection, the sputum was markedly decreased and the patient's health greatly improved. In 1935 he had a recurrent pneumonitis. This involvement

progressed until the entire upper lobe became bronchiectatic. The sputum again became profuse and clubbing of the fingers was marked. This lobe was removed on February 28, 1938. It was shrunken, fibrotic, and ateletatic. In short, it was a fibrotic mass of tissue traversed by tortuous bronchi, the mucosa of which presented rugae and folds due to foreshortening caused by fibrosis of the parenchyma.

Fig. 3.

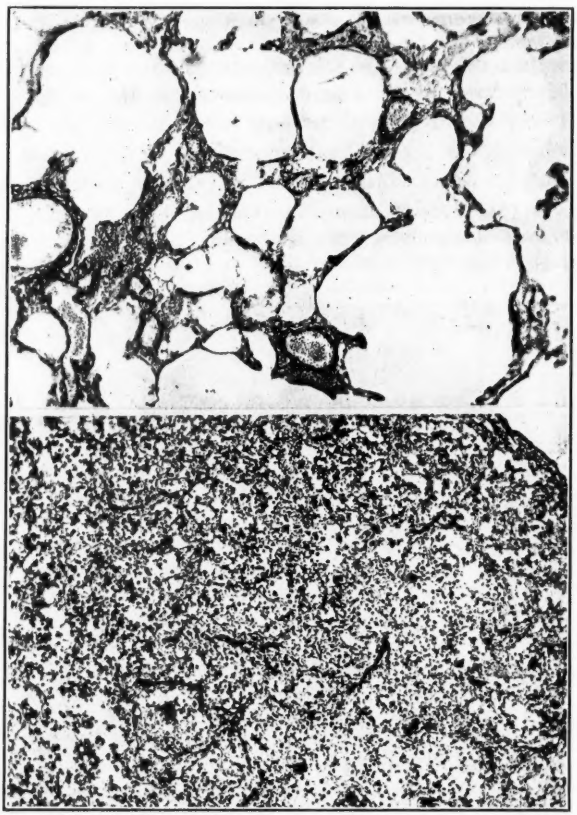

Fig. 4.

Fig. 3.—Emphysema in nonpneumonic portion of lung shown in Fig. 2. Elastic fibers are stained black by Weigert stain.

Fig. 4.—Section of pneumonic portion of lung shown in Fig. 2 showing fragmented elastic tissue fibers (stained black by Weigert stain) indicating that emphysema antedated the pneumonia.

The bronchi were filled with tenacious mucopus. The resection was done through an anterolateral incision. It was technically difficult because of dense adhesions to the lung, holding it in the posterosuperior sulcus posterior to the heart. The operation was well tolerated under cyclopropane anesthesia (right intrabronchial intubation). On the second day he developed a pneumonia of the upper and lower lobes of the right lung. This pneumonia was Type 19 for which, unfortunately, there is no serum. He expired on the fifth postoperative day.

The following is a partial autopsy protocol: The surgical wound was healing satisfactorily; there was no free fluid or pus in the left pleural cavity; the silk pedicle ligatures were intact. The stump of the upper lobe bronchus measured one and one-half inches in length from the carina. The mucosa was inflamed, there was a slight amount of secretion in the bronchus, and a bronchopleural fistula was present. The entire left thoracic space was obliterated, first, by slight flattening of the chest wall, second, by marked elevation of the diaphragm (the dome was at the level of the third rib), third, by the position of the heart, which was lying with its long axis at right angles to the long axis of the body. Its apex was in contact with the chest wall at the level of the second intercostal space and the mid-axillary line. The great vessels were accommodated to the heart's position and were not angulated. This position of the heart was produced by the elevation of the diaphragm and the shifting of the mediastinum to the left, which rotated the heart posterolaterally, fourth, the right lung was enormously enlarged and the gross volume practically equaled the normal amount of pulmonary tissue found in an unoperated chest. The lung "pseudopodia," as Rienhoff calls it, had herniated well beyond the left sternal margin.

Since this was an autopsy on a child, it was hoped that some histologic facts might be demonstrated that would clarify the compensatory dilatation theory advanced by Rienhoff versus the true hyperplasia theory advanced by Bremer,[3] in the growing individual. Unfortunately, from a study of our case, neither theory can be affirmed nor denied. First, we have no preoperative lung tissue for comparison. Second, our patient died from a pneumonia which involved a large part of the remaining lung, and which may have changed the histologic picture. A microscopic study of both the pneumonic and uninvolved sections of the remaining lung, however, showed definite dilatation of the bronchioles as well as of the alveoli, the walls of the latter showing evidence of rupture and fragmentation of their elastic fibers, in other words, the dilatation ordinarily found in lung emphysema.

CASE 4.—H. S., an 11-year-old girl, showed a moderate involvement of the left upper lobe at the time the left lower lobe was removed for marked saccular bronchiectasis. A pneumonectomy was indicated and planned, but the adhesions were so dense that the writers believed it best to remove one lobe at a time. Here again removal of the main nidus of infection resulted in such marked temporary improvement that removal of the upper lobe was postponed. Now, however, at the end of four years, the disease has involved the entire upper lobe, and completion of the left pneumonectomy is definitely indicated. As in the previous case of A. B., the contralateral lung is not involved.

CASE 5.—F. I., a boy 8 years of age was operated on in 1933. The middle and lower lobes were removed for bronchiectasis. The right middle lobe was extensively involved, but the right lower lobe but slightly involved. The result of a two-stage operation has been excellent. There has been normal development, no recurrent pneumonitis, no sputum, and the clubbing of the fingers has completely disappeared.

The electrocardiogram is normal, actual vital capacity is 1580 c.c., calculated vital capacity, 2000 c.c., and tidal air, 300 c.c. The remaining pulmonary tissue shows no abnormality either by x-ray or physical examination.

CASE 6.—L. U., a girl 8 years of age, was admitted to Bellevue Hospital, August, 24, 1934. She had a right lower lobe abscess and bronchiectasis following a tonsillectomy. This patient was treated medically for five months. During this period pulmonary hemorrhage was a serious and recurring symptom. On January 28, 1935, a single stage lobectomy was done as an emergency to control hemoptysis. Convalescence was slow, because of a *Bacillus welchii* infection of the chest wall. The infection was treated successfully with zinc peroxide. (This patient would undoubtedly have died had it not been for the use of zinc peroxide.)

She is practically cured. Her physical development is normal, but mental retardation, frequently found in connection with bronchiectasis, still persists. A recent bronchogram shows no bronchiectasis. The normal angle of the right bronchus is markedly changed to the extent that it is almost a straight continuation of the trachea. The electrocardiogram is normal. Vital capacity and tidal air could not be obtained due to inability of this patient to cooperate.

CASE 7.—E. B., a girl aged 5 years, was admitted to Bellevue Hospital, July 13, 1931, with an acute lung abscess and well-developed bronchiectasis of the left lower lobe following a pneumonia in 1930. She expectorated great quantities of pus, which ultimately proved to be coming from her bronchiectasis and not from the acute lung abscess. The lung abscess was surgically drained in September, 1931, with marked amelioration of the symptoms of bronchiectasis.

As normal development did not follow the operation for lung abscess, and postural drainage was unsatisfactory, the patient was readmitted on January 6, 1936, for lobectomy. While her bronchiectasis was confined to the left lower lobe, x-ray examination repeatedly showed large emphysematous blebs of the left upper lobe. At times, free air in the left apex could be demonstrated by x-ray. On January 18, 1936, a left lower lobe resection was done through an anterolateral incision. The bronchial stump was covered with the normal looking left upper lobe. A bronchial fistula did not develop and convalescence was very satisfactory, although the upper lobe did not re-expand as expected. On discharge three months later, the chest wound was completely healed, but a partial pneumothorax still existed about the left upper lobe.

The child has developed normally since her lobectomy and has no complaints. Recent x-ray and bronchogram studies revealed no bronchiectasis of the upper lobe, which, however, remains in a partially collapsed state. Lipiodol demonstrates a definite bronchopleural fistula opening into the partial pneumothorax. This condition is undoubtedly due to a rupture of emphysematous blebs noted on the x-ray previous to operation. This patient will be carefully followed, but since she is symptom-free at present, no further surgery is indicated. The electrocardiogram showed left axis deviation. Vital capacity was 1360 c.c., calculated vital capacity, 1800 c.c., tidal air, 315 c.c.

CASE 8.—R. G., a boy aged 10 years, was admitted to the Chest Clinic, Bellevue Hospital, January 22, 1937, suffering from a lung abscess of unknown etiology. He received medical treatment for five weeks without improvement, and was then transferred to the Surgical Ward. A portion of the left fourth rib, mid-axillary line, was resected, the abscess exposed and unroofed. It contained 50 c.c. of a semisolid, gelatinous, malodorous mucopus. The stoma of numerous bronchi could be seen entering this emptied abscess cavity. It was packed with vaseline gauze and a bronchocutaneous fistula established. During the ensuing seven months there was marked improvement in his general health. The fistula was then closed with a pedicle muscle flap. Improvement was satisfactory for the following three months, after which expectoration increased and a bronchogram revealed cavitation at the site of the original abscess, involving both lobes of the left lung plus well-developed bronchiectasis. It was evident from physical and x-ray examinations that

the entire lung was atelectatic, fibrotic, and bronchiectatic. Consequently, on February 2, 1938, a single-stage pneumonectomy was done through an anterolateral fourth interspace incision. Grossly, the entire lung was involved. It was firm and contained no air except in a very small area at the extreme apex. The abscess cavity, which had burrowed through to the intercostal muscle, was again filled with the same mucopus.

Dense adhesions at the site of the previous operation and pedicle graft were encountered, otherwise the lung was easily freed. It was removed by the use of a Shenstone tourniquet applied at the hilus. The patient left the table in very good condition. He developed a severe bronchitis of the contralateral lung, and an empyema in the left pleural space. These complications were successfully treated; he is now gaining weight, is in excellent health, and has no expectoration. He still has a bronchocutaneous fistula. This case is included in this report to facilitate a subsequent report on late results in bronchiectasis.

The caption of this report connotes only postoperative results, yet the question naturally arises, what is the result in similar nonoperated cases? Through the courtesy of Dr. Edith M. Lincoln, the results in seventy cases of bronchiectasis treated conservatively in the Bellevue Hospital Clinic for Children are herewith offered. This group includes all types of cases. Some refused indicated surgical treatment. The majority of the cases, however, were suitable for medical care. Seven case histories, of which brief summaries follow, illustrate the clinical features of the various groups.

CASE A.—V. R., a 3-year-old girl, was admitted to the hospital in 1928 because of a persistent cough which followed pertussis at the age of one month. On admission she had marked sinusitis with postnasal drip, cough, and slight clubbing of her fingers. X-ray film showed fibrosis extending from the root towards the periphery of the lung, which a bronchogram revealed to be a saccular bronchiectasis of the right upper lobe. The sinus infection was controlled by irrigation and the lung condition was treated by bronchoscopic and postural drainage. The postural drainage and sinus treatments were continued after her discharge from the hospital. Tonsillectomy done at the age of 7 years was followed by improvement. At present, at the age of 13 years, the treatment still being continued, she is clinically well except for slight cough and sputum aggravated each winter by colds. Her weight is ninety-two pounds and she loses little time from school.

CASE B.—M. J., a girl aged 10 years, was admitted to the hospital in November, 1930. She gave a history of bronchopneumonia in 1929 and since then has had a productive cough. On admission she had a marked sinusitis and a bronchogram revealed fusiform bronchiectasis involving the left lower lobe. The sinuses were treated and postural drainage instituted, but bronchoscopic drainage was refused. The treatment was continued at home, but she continued to have profuse nasal discharge, productive cough, and frequent colds. Operation was advised but refused. She has learned the value of postural drainage, and although she is not entirely well, as in February, 1938, she still complained of pains in her chest, slight cough, and expectoration, she is able to keep her job.

CASE C.—P. S., a 4-year-old girl, was admitted to the hospital in June, 1932, complaining of cough and expectoration. She had just recovered from pertussis and pneumonia. Her admission x-ray examination showed atelectasis of the left lower lobe, and a bronchogram demonstrated a cylindrical bronchiectasis of both lobes. She also had a sinus infection. She refused bronchoscopic examination.

Her sinuses were adequately treated and postural drainage instituted for her bronchiectasis. Under this treatment, combined with bed rest, the lung gradually expanded and she was discharged to the chest clinic. Though intelligent cooperation was lacking, she has slowly improved. She still has a cough and sputum and occasional exacerbations with fever. In February, 1938, she was 10 years old, weighed fifty-eight pounds, and was attending school regularly.

CASE D.—V. B., a 4-year-old boy, was admitted to the hospital in August, 1934. He complained of cough and fever, which had been present since 1932, when he had pneumonia followed by a lung abscess. A bronchogram showed saccular bronchiectasis of the right lower lobe. He was hospitalized for a period of eight months and treated by postural and bronchoscopic drainage. Eight months after his discharge his pneumonitis recurred. In November, 1936, he had a productive cough with fever, infected sinuses, and diseased tonsils. He was lax in carrying out his postural drainage and would not accept treatment for his upper respiratory conditions. X-ray examination revealed an extension of the bronchiectasis to the right middle lobe. He weighed forty-five pounds. Lack of cooperation makes medical treatment valueless in this case.

CASE E.—R. C., a 10-year-old boy, was admitted to the hospital in October, 1932, complaining of cough, nasal obstruction, and blood-streaked sputum. At the age of 18 months a nail had become lodged in his bronchus, and was removed one year later. On admission his sinuses were infected and bronchogram showed a small saccular bronchiectasis of the right lower lobe. With appropriate treatment of his sinus infection, the cough disappeared and when seen in 1937 he was free from signs and symptoms. His weight is now 110 pounds.

CASE E.—R. R., a 5-year-old girl, was admitted to the hospital in May, 1934, complaining of cough, fever, and foul sputum following tonsillectomy in March, 1934. On admission she had persistent cough and moderate fever. Bronchogram showed a cylindrical bronchiectasis of the right lower lobe. She was hospitalized for seven months and treated by bronchoscopic and postural drainage. The productive cough ceased and she was discharged to continue her treatment at home. She continues her postural drainage and is now free from symptoms.

CASE G.—V. B., a 10-year-old girl, was admitted to the hospital in April, 1932, complaining of cough, fever, and loss of weight. She had three recurrent attacks of pneumonia in the left lung. The antra were badly infected. A bronchogram showed a cylindrical bronchiectasis of the left lower lobe. She remained in the hospital three months, during which time her antra were treated and she received bronchoscopic and postural drainage. During the subsequent six years the treatment has been continued in the clinic. Her improvement has been gradual but punctuated by recurrence of symptoms and one attack of pneumonitis. When last seen in October, 1937, she weighed ninety-four pounds and was relatively free from symptoms and signs. She still faithfully practices postural drainage.

The conservative treatment of Dr. Lincoln's seventy cases, illustrated by the preceding clinical histories, indicates that in selected instances bronchiectasis can be treated without operation by the removal of focal infections in the upper respiratory tract and by bronchoscopic and postural drainage continued faithfully for an indefinite period. One patient only is known to have died (brain abscess). Many individuals can continue with their activities with a minimal loss of time on account of recurring exacerbations, and some may even attain a clinical cure. Normal growth and development are quite possible. In several in-

stances patients have reached adult life; one, a woman, has married and assumed adult responsibilities. They must, however, continue postural drainage. The reason for this is well demonstrated by the late Dr. Jacobaeus.[4] He has shown on his bronchospirometric curve that when a lung is diseased the act of coughing is done entirely by the sound lung and that secretion from the diseased lung can be voluntarily raised only after it has reached the tracheal bifurcation. Hence, there is the necessity for frequently applied and long-continued postural drainage.

DEDUCTIONS AND SUMMARY

In this series of ten patients, a single stage pneumonectomy was done in one, complete pneumonectomies in two, the upper lobe removed at later operations. The right lower and middle lobes were removed in one. A lobectomy was done in the remainder of the series.

1. When the entire pathologic pulmonary tissue has been removed, cure has resulted.

2. Where the lower lobe has been removed, in patients with only slight involvement of the upper lobe, the disease has progressed, necessitating an upper lobectomy.

3. Pneumonectomy in stages where indicated, is feasible.

4. Postural deformities have not occurred.

5. It has not been necessary to do a phrenic resection or crushing to obliterate the space.

6. Thoracoplasty has not been necessary in patients with pneumonectomy, as proved by x-ray examination and, in one case, by autopsy.

7. Elevation of the diaphragm and shift of the mediastinum has caused no visceral or circulatory disturbance.

8. Electrocardiogram is essentially normal in all these patients. No cardio-respiratory disturbance has been noted, even in the case of A. B., when the heart was in a transverse position.

9. Neither bronchospirometric curves, oxygen, nor carbon-dioxide content of alveolar air were determined, but clinically there was no disturbance in the pulmonary interchange of gases. These patients are able to undergo violent exercise with the normal amount of fatigue.

10. Histologically, emphysematous dilatation of the remaining lung was found in the one autopsied case.

11. When unilateral diseased lungs were operated on there was no spread of the contralateral lung.

12. Physical development has been normal.

13. The normal angle between the stem bronchus and the trachea has become increased on the uninvolved side and decreased on the operated side. This caused difficulty in passing a bronchial catheter in the stem bronchus of the uninvolved lung.

14. Medically treated patients do surprisingly well, but endless, intelligent, methodical attention is required.

15. Mortality in nonoperated patients, at least up to the age of 20 years, is not great. Recurrent pneumonitis and metastatic brain abscess account for most of the deaths.

16. Moderate or even advanced bronchiectasis is not incompatible with fair longevity.

17. Social ostracism and mental complexes are common handicaps.

If the writers may be permitted to speculate on the future of surgery of bronchiectasis, we predict that its surgical relief will be very much more frequent during childhood, for soon the pediatricians will not hesitate to refer these patients to the thoracic surgeon for cure and thereby greatly reduce the incidence of bronchiectasis in adults

This will come about because (1) surgery is the only cure; (2) children withstand the operation better than adults; (3) it will obviate complications; (4) it will prevent deformities; (5) it will prevent ostracism of these patients.

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DISCUSSION

DR. CHARLES W. LESTER, NEW YORK CITY.—I would like to discuss a little more in detail the third case which Dr. Bohrer showed, the case which came to autopsy.

The sketch which he showed on the screen was of course made at autopsy. At operation essentially the same condition was present, because the remainder of that left lung, the left upper lobe, was shrunken, atelectatic, bronchiectatic, and smaller than my fist. The right lung was doing all the work and was pushed over pretty much in the position in which it was found at autopsy. If you remember the picture Dr. Rienhoff showed before this Society in 1936 you can see the similarity between that picture and the one which was just shown here.

In Dr. Rienhoff's case, although the gross picture was very much the same as ours, the microscopic picture was quite different. It showed a "compensatory dilatation of the alveoli." This condition has also been noted by Laennec and by various other foreign pathologists and clinicians since his time, but was brought to our attention more recently by Dr. Rienhoff. In that state the alveoli are enlarged but the walls are intact, the elastic fibers are intact, and, in short, you have a lung which is large and taking the position of the other lung so far as work and location go and really a functionally good lung.

When you look at the slide of this lung from our autopsied case, it is perfectly evident that we did not have a functionally good lung. The lung is riddled with emphysema, and the emphysema existed before the terminal pneumonia, because the ruptured elastic fibers can be seen in the section taken through the pneumonic area.

I do not think that this is a new condition by any means. I believe that the lung in the first place was in the same sort of condition that Dr. Reinhoff has described, that is, it had a compensatory dilatation or hypertrophy, as Laennec called it. This boy had frequent attacks of pneumonitis, off and on over a period of several years, one at least severe enough to confine him to the hospital, and several others which confined him to bed at home.

James Alexander Miller has recently shown that there is a definite sequence of events in chronic pulmonary conditions. You have a chronic infection that is followed by fibrosis and the fibrosis in turn is followed by emphysema. This lung showed, in addition to the emphysema, in other portions of the slide, a very marked fibrosis, and I think that that is what happened in this case.

The lesson to be learned from this is that bronchiectasis treated over too long a time unsuccessfully by medical means will bring about emphysema which gives a bad lung because the emphysematous lung has lost its self-cleansing powers and is more liable to infection such as the postoperative pneumonia which caused the death of this individual.

This would mean that when medical treatment has proved unavailing, when the patient is no longer improving, he should be referred for surgery right away and not temporized with for a period of months or even years, as so often happens now. It also means that when more than one lobe is involved the entire diseased area should be removed at one time, if possible. If, however, that proves too much of an operation, it is feasible to do it in stages, as happened in the first case of Dr. Bohrer's. However, between the stages the child should be very carefully watched, should be tended almost like a hothouse plant, and should be protected from respiratory infection or anything which would set up a condition leading to emphysema.

DR. L. J. LEAHY, BUFFALO, N. Y.—I would like to comment very briefly on Dr. Bohrer's second case, in which he left behind residual tissue which remained viable.

I think it has been shown that even though the pulmonary circulation is cut off at the hilus, unquestionably the remaining lung can remain viable. If you recall Dr. Beye's case, which was mentioned by Dr. Bohrer, he very stoutly ligated the hilus of the lower lobe and at a later operation found blood vessels as large as a lead pencil which had come through the pleura from the bronchial circulation.

Also there is the report of a case by Dr. Windsberg, in which he ligated the hilus and cut it across, thinking he had performed a pneumonectomy and later found at autopsy three years later that the lobe had remained behind and survived even after division of the hilus.

The successful cases, like Dr. Sauerbruch's, Dr. Nissen's, and Dr. Haight's, all make mention of the fact that the pleural cavity was free. We recently had a case in which a girl with bronchiectasis had been perhaps improperly treated before she came to our hands, with artificial pneumothorax, and when we saw her she required extensive extrapleural thoracoplasty to obliterate the chronic empyemic cavity; this was successfully done. A destruction of the lower lobe by repeated cauterizations was done, but this procedure did not accomplish its purpose, and we attempted a pneumonectomy. The adhesions were so dense it seemed as if the lower lobe were part of the chest wall, so we contented ourselves with a very tight rubber tourniquet about the hilus and packed the wound, hoping there would be a sequestrum of the lung. The upper lobe did come through the wound in about ten days; a bronchial fistula appeared, but later closed; her sputum stopped entirely, and she was around for about eight weeks when she developed a bronchopneumonia on the other side and died. The lower lobe was still present, embedded in dense adhesions.

I think, in addition to the dilatation of the bronchial vessels from inflammation and ligation of the hilus, definite changes take place within the lobe in the nature of a carnification, so perhaps the operation of applying a tourniquet about the hilus and expecting a sequestration is not sound in the presence of dense adhesions. Surgical dissection should be done and the lobe and lung removed at once.

DR. EDWARD D. CHURCHILL, BOSTON, MASS.—I should like to confirm Dr. Bohrer's report regarding the accelerated growth, the gain in weight and the improvement in general well-being that follows lobectomy for bronchiectasis in children, and this is true even though the child is apparently not very sick. Children are likely to be peculiarly inarticulate about their symptoms and consequently the symptoms are likely to be minimized by their doctors unless gross objective signs, such as foul or profuse sputum, are present, or unless the child has unusually observant parents.

A child with bronchiectasis is often proud of the fact that he can spit. I had one patient who said as a child she used to "spit for the other children and tell them it was poison." It was an achievement that delighted them. This attitude is apt to collapse with adolescence and then these patients really begin to get into serious psychologic difficulties as a result of their disease, even though the foul sputum is lacking.

I think we need more statistics such as Dr. Bohrer presented in regard to bronchiectasis treated over a long period of time by nonoperative measures.

I should like to ask Dr. Bohrer if these seventy cases were all demonstrated to have bronchiectasis by lipiodol, which I think is the only certain way of establishing the diagnosis. Pediatricians and sometimes radiologists are particularly prone to place a label of bronchiectasis on some children with a "postnasal drip" and a cough, purely because of the increased markings in the basal lobes of the lung. We cannot accept that type of case as bronchiectasis. I assume Dr. Bohrer's cases were all proved by lipiodol.

Dr. Lester has brought up a very interesting point. We have to admit that bronchiectasis, under observation, progresses very slowly if at all. He has called attention, however, to another change, an irreversible destruction of the remaining lung tissue that is ultimately as fatal as the bronchiectasis—fibrosis and emphysema due to the constant bombardment of the normal lung by bronchogenic infection causing focal areas of pneumonitis. This particularly should be borne in mind in judging operability in a proposed program of bilateral lobectomy. We must be sure not only that there is no bronchiectasis in the upper lobe, but that there is not this loss of function due to fibrosis and emphysema.

DR. ETHAN FLAGG BUTLER, ITHACA, NEW YORK.—Dr. Bohrer is to be congratulated for two reasons: first, his excellent series of cases, second, his close relationship to a very cooperative pediatric service in Bellevue Hospital. Dr. Edith Lincoln, Director of this pediatric service, has discussed her work before the New York Society for Thoracic Surgery and, in my opinion, has presented strong evidence that intelligent, long-continued medical care during the initial phases of bronchiectasis (her diagnoses were supported by convincing bronchograms) could prevent the later development of the type of disease with which the surgeon is familiar. When one realizes that Dr. Bohrer's surgical cases have come from this same pediatric service one has to concede a liberal attitude and a careful differentiation between cases for which medicine holds no prospect of relief and those for which a medical regime is worth while. We surgeons can profitably listen at some future time to Dr. Lincoln's opinions on the early evolutionary stage of bronchiectasis in young children.

In my own work five children of 13 years or less have come to lobectomy. Three received lobectomy because of pathology resulting from long-retained foreign bodies. Two of these three children survived; one of them in spite of a gas bacillus infection. The organisms probably gained access with the foreign body, a "timothy" head.

DR. J. J. SINGER, LOS ANGELES, CALIF.—Dr. Churchill's remarks that he thought the Association had settled empyema and bronchiectasis brought something to my mind that is rather acute. The surgical branch of this Association does not see the cases of bronchiectasis until everything has been done medically, and a great deal of discussion between the doctor and the patient has been had before we can convince the parents that the operation might do them some good. Now it is an interesting fact that in bronchiectasis really very little has been done that could possibly be called a cure, other than lobectomy. The very detailed medical care suggested by the writer that has been used in the seventy-odd cases at that particular hospital is not available, I would say, to 90 per cent of patients, or if available, the symptoms sometimes are not sufficient for them to use it.

DR. ROBERT M. JAMES, TORONTO, ONTARIO.—It might be interesting to mention a little girl who has been of great interest to Dr. Thurstone and myself. This was a child between 19 and 20 months of age who weighed fifteen pounds. The illness began with whooping cough at 6 months of age. The child had an extensive bronchiectasis of the right upper lobe with a great deal of very foul sputum. It was impossible to tell how much because the sputum, of course, was swallowed.

She was operated upon five years ago, a one-stage upper lobectomy done. There was no particular difficulty despite the sort of miserable little patient we had. She stood up well to the operation, except for the difficulty of keeping the bronchus clear of secretion. The amount of sputum was tremendous.

That child is now between 6 and 7 years of age and is healthy and normally developed.

I think Dr. Churchill mentioned something that is very important. I do not believe that the question of bronchiectasis is settled, indeed, scarcely more than begun. In Toronto at the moment we are running a combined medical and surgical clinic in an effort to find out what cases of bronchiectasis should be operated upon. One is already impressed with the fact that many patients may live to middle life and beyond without any particular advance, or, so far as one can tell from history, increase in symptoms. There are others who suffer from a progressive type of disease. There is a great need for combined medical and surgical clinics conducted not for a short time, but, as we hope, to carry this over a period of many years, in order to find out exactly what patients should be operated upon and what patients should not.

DR. POL N. CORYLLOS,* NEW YORK CITY.—In the discussion of the excellent paper of Bohrer and Lester, pneumothorax and deep x-ray treatment were mentioned in the management of bronchiectasis. I shall say a few words on these two methods of treatment.

I consider that artificial pneumothorax may have disastrous affects on bronchiectasis without offering any serious hope for cure to the disease. I performed last year a pneumonectomy on a patient with bronchiectasis of both lobes of the left lung. The patient was a white woman, a nurse, in Binghamton, N. Y., under the care of Dr. Mark Williams. Pneumothorax had been induced by another doctor and was followed by empyema which was drained. This left pneumonectomy was technically extremely difficult. The patient has recovered but after a long and stormy postoperative course. It certainly would have been more reasonable to have

*Deceased.

this operation performed two years previously without exposing the patient to the complications which had followed a useless pneumothorax.

Lately, I had a sad experience regarding deep x-ray treatment. I had operated three years ago, on a white girl, aged 15 years, at the Metropolitan Hospital of the City of New York. This patient had bronchiectasis of both lower lobes. I resected the lower lobe of the left side, and the patient had an uneventful recovery. For six months there was a marked improvement on the right side as this occurs occasionally. Gradually, however, cough and expectoration had markedly increased. I thought it advisable to have this patient submitted to deep x-ray treatment before proceeding with a lobectomy on the right side. She had two series of treatments without any improvement. After the second series she had a very acute exacerbation of the anaerobic infection of the right lower lobe and died within a few days with symptoms of gangrene of the lung. No autopsy could be obtained.

DR. EVERETT C. DRASH, CHARLOTTESVILLE, VA.—I am glad this question of x-ray therapy for bronchiectasis has arisen. There have been a number of reports in the literature, apparently with excellent results. We have seen one patient with a very similar reaction to that reported by Dr. Coryllos, in which, a few hours after a rather heavy dose of x-ray therapy, the patient began to complain of severe dyspnea, and by late evening, within six or eight hours, the patient was quite cyanotic, had very difficult respiration, and was really in a state bordering on pulmonary edema.

It seems to me that even if these early severe reactions are avoided, the principle of x-ray therapy for bronchiectasis is wrong. It is obviously impossible to sterilize these severely infected areas, and it is certain that infection will recur after the effect of the x-ray has subsided. It seems to me there is another effect which is bound to occur that may be highly dangerous; that is, that after the x-ray effect has subsided and the fibrous tissue reaction begins to occur, most patients will probably develop bronchial stenosis and extensive fibrosis which will render the condition worse than it was originally. I think that x-ray therapy is a poor principle for the treatment of bronchiectasis.

DR. JOHN V. BOHRER, NEW YORK CITY.—Permit me to thank the members of the Society for their generous discussion of this paper. I will make my remarks brief.

In regard to Dr. Churchill's question, I will state that these medically treated cases have all been bronchogrammed. This group has been very carefully studied by Dr. Lincoln and her associates. They have been divided into three classes; those in the advanced stage where the bronchiectasis consists of large pockets, the intermediate stage, and those where the diagnosis can be made only by very careful examination and bronchogram.

We all believe that the first group is essentially surgical and that the third group, those just beginning, is definitely not surgical. But, there is considerable conflict existing about the intermediate group, the medical side being more conservative. There is no question, however, about the improvement of these medically treated cases. The only point that comes to my mind is whether the delay in operating will be disastrous. Some of them will come to operation later and it will be difficult, and perhaps impossible, to get as good results as could be obtained in the early stages.

Concerning Dr. Singer's remarks on the disadvantage of bronchoscopy, I would say that the results are much better in children than in adults. Postural drainage and bronchoscopic drainage, until the infection has subsided, are of distinct advantage in childhood. I have seen a number of moderately involved cases show great improvement. Growth in the child has much to do with the result. The dilated bronchus is gradually corrected by growth. The French writers make quite a point of this fact.

THE MECHANICS OF PULMONARY ABSCESS, PULMONARY GANGRENE, AND BRONCHIECTASIS, WITH A SUGGESTED METHOD OF TREATMENT

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CHICAGO, ILL.

PULMONARY suppuration, as evidenced by the syndrome of gangrene, abscess, or bronchiectasis, may be looked upon as a distinct type of pneumonia in which a specific group of conditions and organisms is generally found. The conditions involved in the production of this pneumonia may be trauma, pulmonary embolism with coincident or subsequent infection of the resulting infarct, aspiration through the trachea into the lung of foreign bodies such as objects commonly or accidentally present in the mouth, blood and mucus from the nasopharynx, or gastric contents that are accidentally spilled into the lung. The organisms most commonly found during the acute as well as the later stages of this lesion are of the fusospirochete group and include the spirochetes of Vincent, the fusiform bacillus, pyogenic cocci, and at times the influenza bacillus and the diphtheroids. Other organisms have also been mentioned as etiologic agents.

Through the work of Pilot and his co-workers[1] we note that the fusospirochetes are present in specimens of lungs with abscess or gangrene. Maes[2] gives credit to Castellani[3] as being the first to recognize, in 1906, spirochetal gangrene as an entity. A number of others, namely, Kline,[4] Rona,[5] Rothwell,[6] Peters,[7] Dick,[8] Nolf,[9] Parof,[10] Thomson,[11] and Weil[12] have written papers emphasizing the association of the fusospirochetes to pulmonary suppuration. The experiments of Lemon[13] clear up a number of important factors in the etiology of the aspiratory type of abscess. He has found that unless the dog is anesthetized no aspiration occurs into the lung. He further states that the fluid preceded the particulate matter, and that when the head of the animal was 28.75 cm. lower than the feet, aspiration did not occur in his animals. Hedblom[14] called attention to the fact that pulmonary infection in man following aspiration must depend largely upon the nature, or the virulence of the infective organisms, or both. From the statements of Lemon and Hedblom we must assume that in pulmonary suppuration, aspiration and anesthesia are of great significance, and also the type as well as the virulence of the organism. That blood finds its way into the trachea in patients undergoing tonsillectomy under general anesthesia has been shown bronchoscopically by Myerson.[15] He has brought out the importance of the abolition of cough reflex in aspiration. Corper[16] has shown

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Read before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

that aspirated blood and fluids reach the alveoli very readily. Coryllos and his associates,[29] among others, have emphasized the importance of bronchial obstruction and its relation to atelectasis, which may aid in the growth of the fusospirochetes, and result in pulmonary suppuration.

David T. Smith,[17] Crowe and Scarff,[18] Allen,[19] Hedblom, Joannides and Rosenthal[20] have written on the experimental production of aspiratory suppuration in the lower animals. These papers bring out the fact that to produce an experimental aspiratory abscess it is necessary to induce a local anesthesia of the pharynx, or a sufficiently deep general anesthesia to abolish the cough reflex; it is also necessary to introduce either scrapings from the teeth and the gums of patients with pyorrhea, or to introduce into the lung the sputum of patients with pulmonary suppuration; they mention the fact that a larger percentage of abscesses is produced if these scrapings or the sputum are mixed with fresh or freshly clotting blood, and finally that the aspirated contents should remain in the lung sufficiently long to allow coagulation of the blood in the finer ramifications of the bronchial tree, and the alveoli.

One must not lose sight of the fact that pulmonary embolism with coincident or subsequent infection of the infarct is also an important factor in the etiology of this lesion. The experiments of Holman, Weidlein and Schlueter[21] and also Cutler and Schlueter[22] have certainly proved the importance of this etiological factor. However, after the infection has set in, the lesion follows a similar course as in the aspiratory type of lesion, and depending upon the degree and severity of involvement the end result is manifested either as a gangrene, or as a pulmonary abscess, or as a bronchiectasis.

A brief description of the lung structure may aid in the better understanding of the mechanics of pulmonary suppuration. In a stereomicroscopic study of the lung (1928 to date) I have found[23] that the lung may be looked upon as a multisaccular organ which is covered by a thin watertight and airtight layer of pleura (Fig. 1). The saccules are multiform and are arranged in clusters much like the clusters of grapes. These saccules are the alveoli and are grouped about the terminal bronchiole which has a characteristic circular staircase appearance with bulbous grooves along its outer walls. The terminal bronchiole is known as the alveolar sac in the anatomic classification of Miller[24] because its tubular phase is not evident in the ordinary paraffin section or reconstruction study. The walls of the alveoli are very vascular and carry a large number of one-cell capillaries through which the diffusion of gases between the alveolar air and the blood takes place. Because of the large amount of elastic tissue present in the interstitial layers of the lung, the alveoli act much like toy balloons by showing a natural tendency to contract. The air gets into these saccules only by virtue of an increased negative pressure outside of the lung so that air rushes in through the unobstructed trachea and bronchi. The natural tendency

of the alveoli to shrink with increasing contraction of the healing interstitial tissues causes the chest cavity to modify itself sufficiently to allow for diminution of total volume of the lung.[28] This tendency to contraction of the lung and the modification of the chest wall is of great significance in the treatment of suppurative pneumonia.

The changes occurring as a result of pulmonary inflammation are as follows: In the presence of an irritating agent, be it physical, chemical, bacterial, aspiratory or embolic, we have noticed[25] in the lung the same changes that occur in inflammation of any tissues or organs. These consist of hyperemia, stasis, and exudation. They are followed by changes showing destruction or repair in the inflamed tissues. Because the alveolar walls are very thin and are surrounded by air spaces, it is obvious that any exudation will naturally find its way towards the point of least resistance, and this is in the direction of the contents of

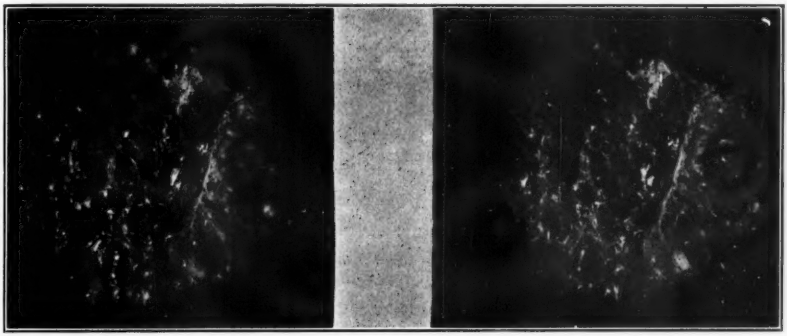

Fig. 1.—Section of a normal lung (×12) distended to its normal volume and dried. The alveoli are seen in their relation to the interlobular septa. (For a detailed view attach picture to cardboard and adjust for stereoscopic vision.)

the alveolar saccule. The alveolar sacs become filled with exudate and as the exudation increases the terminal bronchioles as well as the smaller bronchi become filled because the air present in the alveoli is displaced toward the trachea. During this stage one may notice a dry, irritating, nonproductive cough, and as the exudate assumes a more viscid consistency the cough becomes more and more productive. This stage may be considered as the endogenous type of alveolar drowning. When fusospirochetes are present in the mucous secretions of the bronchi, these organisms may be caught in the exudate. Now that anaerobic conditions are established, the growth of the fusospirochetes becomes possible. Putrefactive changes then are likely to occur and suppuration of the lung may follow. If the pulmonary inflammation is extensive enough to involve a large portion of the lung, a gangrene of the lung may result. If, finally, only a very small area or multiple small areas are involved, particularly in the base of the lung, multiple small dissociated abscesses may occur which are not likely to drain satisfac-

torily and thus lead to the destruction and subsequent chronic distortion and dilatation of the corresponding bronchioles. If the true nature of the lesion is not recognized during the acute stages and proper treatment is not instituted, destruction of lung tissue may occur, and when a bronchial communication is established, varying amounts of foul-smelling sputum are coughed up. The clinical findings depend entirely on the coincident modification of pathologic changes and also on the circumstances existing in each individual case.

In the aspiratory type of pulmonary suppuration a similar set of findings can be noted with the only difference that the exudate is exogenous and not endogenous. In this type of lesion it is just as easy for the aspirated material to find its way into the smaller bronchioles and alveoli and in this way entrap itself there particularly if it is heavy enough or if there is any tendency on the part of the material to produce fibrin, or if it is difficult or impossible for the patient to cough up this material.

Unless one is on the alert for this type of lesion, the patient may easily be labeled as a case of ordinary pneumonia and the proper treatment is not instituted until liquefaction and suppuration have occurred. This error is particularly likely to occur if no roentgen study of the lung is carried out during the early stages of this syndrome. Another factor in causing an error in the proper diagnosis of the lesion in the earlier stages is the neglect to look for fusospirochetes in the sputum. In every case of clinical pneumonia one should examine for fusospirochetes in addition to the routine study of the sputum for pneumococci and tubercle bacilli.

When conditions become favorable for healing, the exudative changes are first to disappear. There is a varying degree of fibrous tissue that develops and this has a tendency to contract. In so doing the contracting fibrous tissue causes the mediastinum, or the diaphragm, to become displaced toward the pathologic side and the upper three or four ribs to sag downward.[28] If the wall of the abscess is thin and drainage of the abscess is satisfactory, the contracting fibrous tissue diminishes the size of the cavity until it completely disappears (Figs. 2 and 3). In this stage the patient may complain of considerable irritating, nonproductive cough which gets worse at night or during the presence of certain atmospheric conditions. At the suggestion of Dr. Petersen[26] we have watched the relation of atmospheric pressure and temperature in relation to this and a number of other pulmonary findings. We have noted that the patients are likely to do badly when the atmospheric pressure is about to drop from a high peak level to a lower level. Under such conditions we have found that patients with all types of pulmonary lesions complain of pains more severely, they cough oftener and harder, and have a tendency to pulmonary hemorrhage. We have seen epi-

demics of pulmonary hemorrhages in which we know of no other direct causative factors except the atmospheric changes.

The fibrous tissue of the involved area continues to contract as long as the drainage from the lung is maintained. This is done by loosening and coughing up the small mucous plugs present in the smaller bronchi. The scar finally reaches a point where it is visible on a radiograph only as a line extending from the hilum towards the periphery.

If the lesion is in the pulmonary bases one must take greater precautions to remove mucous plugs from the smaller bronchi. Areas distal to such plugs are likely to undergo suppuration and cause dilatation of the bronchi. Injections of iodized oil at intervals of seven to ten days not only aid in the study of the bronchial tree but also help in the loosening of mucous plugs from the bronchi and thus promote drainage. Dr. Schlack[27] has been able to cause a complete disappearance of bronchial dilatations by instilling iodized oil into the lung. Naturally one must always be cognizant of the dangers of complications associated with this procedure.

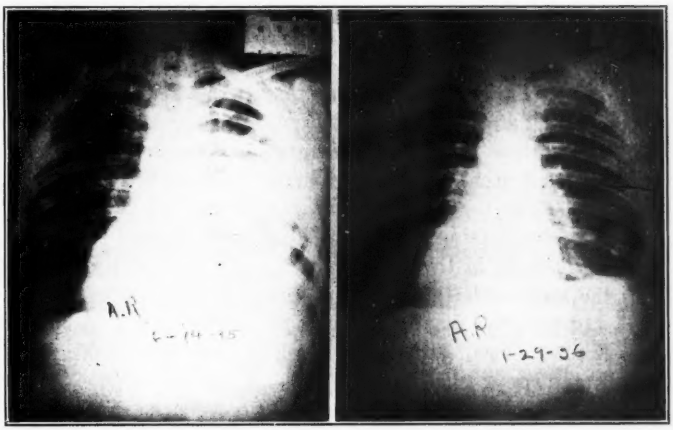

Fig. 2. Fig. 3.

Fig. 2.—Pulmonary abscess with beginning central necrosis.
Fig. 3.—Pulmonary abscess. Disappearance of pathologic evidence of previous lesion. (Arrow points to site of previous lesion.)

SUGGESTED TREATMENT

Based on our present knowledge of the etiologic factors and various phases of the lesion in suppurative pneumonia, we suggest the following treatment which has proved successful in our hands.

1. In patients who have a tendency to postnasal drip or are about to have an operation on the nasopharynx, or the mouth, a definite effort should be made to clear up the gums from pyorrhea alveolaris, and remove all tartar from the teeth.

2. Patients who are to have an operation about the nose and throat should be kept in a position that is least likely to result in aspiration of blood into the lung, and particularly so if it becomes necessary to induce a local anesthesia of the nasopharynx or a general anesthesia deep enough to paralyze the cough and swallowing reflexes.

3. Patients having a clinical picture of pneumonia should be studied by means of x-rays and special sputum examinations to determine the presence of fusospirochetes or an evidence of beginning liquefaction showing in the x-ray film.

4. With the earliest indications of a suppurative pneumonia the patient should be given some form of the arsphenamine group of drugs till the sputum shows no more spirilla of Vincent. We give 0.3 to 0.6 gm. of sulfarsphenamine intravenously at three- to four-day intervals.

5. Bronchial obstruction may be relieved by increasing the moisture in the patient's bedroom and also by direct inhalation of gomenol. We use one teaspoonful of gomenol in eight to sixteen ounces of normal salt solution for vaporization. This inhalation is continued until the patient notices some engorgement in the conjunctiva or the nasal mucous membrane, and is not repeated till the engorgement has cleared up. This engorgement causes a sufficient amount of hyperemia in the bronchial mucosa to allow dry, gummy, mucous plugs to be loosened and coughed up. As soon as there is no more sputum, the gomenol inhalations may be used only at three- to seven-day intervals. More frequent use of this type of treatment usually leads to an acute chemical bronchitis and may result in a serious hemorrhage from the lung. We have found that gomenol is more soothing to the bronchial mucosa than oil of cajeput, oil of eucalyptus, oil of thyme, or compound tincture of benzoin.

6. As the lung heals there is a tendency on the part of the lung to contract and pull the diaphragm upward. Where there is a tendency towards fibrosis, showing in the x-ray film, and the diaphragm shows no tendency to rise, a phrenic nerve crush may assist in giving a chance to the fibrous tissue to contract more readily and result in more rapid healing.

7. Care should be taken to diminish the tendency to cough as soon as the sputum has been reduced to about 30 c.c. per day. This precaution will prevent pulmonary hemorrhage and bronchiectasis resulting from the stretching of the scar tissue and the weakened bronchi in the involved area.

8. We have had two fatalities resulting from an acute extension of a small abscess into the whole lung by the use of artificial pneumothorax. Apparently the air pad outside of the lung causes a kink in the proximal portions of the bronchi. Such a distortion or stenosis may cause a greater tendency towards anaerobic conditions in the lung. It may also cause infected exudates to spill into uninvolved pulmonary areas, thus producing an extension of the lesion.

We also had a death from an acute suppurative pleuritis resulting from artificial pneumothorax collapse.

9. We recommend the use of bronchoscopic drainage in cases in which we cannot obtain a satisfactory drainage of the lung by means of inhalations of gomenol, or iodized oil instillations.

10. We recommend the use of iodized oil in chronic basal suppurations. The routine used by Dr. Schlack of Oak Forest, Ill., has given us the best results.

11. If the abscess wall is too thick to allow for contraction and closure of the abscess, and does not respond to conservative forms of drainage, the patient should be subjected to surgical care.

The aid and support of the Board of Directors, Municipal Tuberculosis Sanitarium, Chicago, that made this study possible are gratefully acknowledged by the author.

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DISCUSSION

DR. JOSEPH WEINBERG, OMAHA, NEB.—I wish to call attention to one point which Dr. Joannides has not considered, and that is the importance of mechanical factors in the production of infection in the lung. A year ago I reported in the JOURNAL OF THORACIC SURGERY some experiments in which I think it was quite conclusively shown that organisms, while an important factor, are not nearly as important as the mechanical factors in the production of bronchiectasis. Bronchial occlusion with its consequent atelectasis will produce a violent and persistent infection without the introduction of outside organisms. But if one introduces organisms without otherwise disturbing the normal bronchial tree, it is almost impossible to produce infection. While the introduction of highly virulent specific organisms might accelerate or increase the severity of the infection, the fact remains that they are less important than the mechanical factors. In this regard one should always keep in mind the possibility of the presence of an occluding foreign body in connection with bronchiectasis or other pulmonary suppuration.

Another important point is that it is necessary to differentiate between the various pathologic types of bronchiectasis in deciding whether surgical or nonsurgical treatment should be employed. For example, a moderate cylindrical bronchiectasis is obviously not a case for surgical procedure. I have seen sections of material taken from lobectomy cases in which the pathologic changes were certainly of too moderate a degree to warrant lobectomy. In my opinion, the only cases which should be subjected to this radical procedure are those with extensive saccular suppurative changes, even though these are less promising for the surgeon from the standpoint of recovery.

I wish to say a few words about the use of iodized oil. Some years ago Dr. Singer called attention to the danger of using too large a quantity of iodized oil in the lungs. If introduced in excessive amounts it may cause damage by plugging too great a number of the bronchi and the bronchial tubes, and this may result in more harm than good. Also, the type of oil which is used is very important. In my experience the chlorinized or brominized oils have proved unsatisfactory because of their irritating effects, and lipiodol has been the most satisfactory, probably because of its stability.

DR. JOHN W. STRIEDER, BOSTON, MASS.—I should like to call attention, if I may, to a small group of cases which were illustrated to some extent in Dr. Joannides' slides, and in which we have found x-ray therapy of value.

This small group of cases has a diffuse pneumonitis with or without cavity in the early stage of disease, which has failed to do well with ordinary conservative measures and in which, either because of the early diffuse phase of the disease or because of other reasons, such as age or the general condition of the patient, operation is not immediately indicated.

At the Boston City Hospital and Massachusetts Memorial Hospital we have had five in this group, all of which were observed with conservative measures for from two to three weeks, during which time the lesions have grown worse or have not improved both clinically and by x-ray. To these we have given small doses of 100 R. in front and in back at weekly or semiweekly intervals and have been very much gratified with the result in prompt or reasonably prompt resolution of the process. I hesitate to advocate this as a routine measure but I do think it has a place of application in a small group of cases during the period of conservative treatment

when they are acute and when operation is not indicated. We have observed twenty-eight cases of pulmonary abscess during the past year and a half at those two institutions, and twenty-two of them came to surgical drainage of the abscess. Of the remaining six, these five were treated by x-ray with gratifying results.

DR. JEROME R. HEAD, CHICAGO, ILL.—The second factor is alcoholism. Deep intoxication is not different from deep anesthesia. If a person with bad teeth becomes deeply unconscious and lies on his back, as he usually does, the chances that he will aspirate oral secretions are excellent. Attempted suicide by overdoses of the barbiturates operates in the same manner and is a common cause of lung abscess.

That most lung abscesses are caused by aspiration of oral secretions is further suggested by the fact that most of them are located in the upper part of the lower lobe directly posterior to the hilus. This is the portion of the lung into which lipiodol flows if it is injected through the bronchoscope while the patient is lying on his back.

DR. RALPH B. BETTMAN, CHICAGO, ILL.—I would just like to report an interesting observation which will fit in here. For many years I was in charge of a dog surgery class at Northwestern University. The procedure of the group was to anesthetize the dog and then have the students perform some type of surgical operation, and at the end of the operation to sacrifice the dog.

For many months, at every experiment we put a few drops of gentian violet in the back of the dog's mouth and at the end of the operation we opened the lung, and in practically every instance both lungs were stained deeply with gentian violet so that in every anesthetic the secretion from the mouth penetrated to the lungs.

DR. MINAS JOANNIDES, CHICAGO, ILL.—I wish to thank the gentlemen for discussing my paper and the kind remarks they have made.

In closing I wish to emphasize again the importance of early diagnosis, and proper drainage of the lung through the bronchial tubes.

Thank you.

ASPIRATION BIOPSY OF TUMORS OF THE LUNG

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THE first diagnostic puncture of the lung was reported by Leyden,[1] in 1883. He used the method for the purpose of obtaining microorganisms from a pneumonic lung.

Menetrier,[2] in 1886, made a diagnosis of carcinoma of the lung by microscopic examination of tissue aspirated during the puncture of an infected tumor of the lung. Talamon,[3] Patella,[4] Tchistovitch,[5] and other early observers mention the use of diagnostic puncture of the lung, with particular reference to bacteriologic diagnosis. Hellendall,[6] in 1899, presented a comprehensive report, "Ein Beitrag zur Diagnostik der Lungengeschwülste," in which he credits Krönig, as being the only previous observer to establish a histologic diagnosis of a tumor by probatory puncture. Hellendall reports two cases in which an ante-mortem diagnosis of sarcoma of the lung was established by means of probatory puncture of the tumor and microscopic examination of the aspirated material. In both cases the ante-mortem diagnosis of sarcoma of the lung was confirmed by autopsy. The first case was one of primary round cell sarcoma of the lung; the second case was a sarcoma of the lung, metastatic from a primary fibrosarcoma of the back. Fränkel,[7] 1896, Sokolowsky,[8] 1898, and Jacobson,[9] 1899, discussed the diagnosis of tumors of the thoracic cavity and mentioned probatory puncture. Quinke,[10] also Ehrlich,[11] reported the recovery of carcinoma cells from pleural exudates.

In 1900, Gumprecht[12] mentioned the rarity and lack of material in the literature concerning the complications of thoracentesis and exploratory puncture of the lung. Fatal complications have since been reported in the literature.

Naunyn,[13] in 1889, Waldvogel,[14] 1906, Bönniger,[15] 1907, Metzlar,[16] 1915, and Stähelin,[17] in 1919, reported cases in which death occurred, generally due to perforation of intercostal arteries and atheromatous vessels within the parenchyma of the lung. With the exception of Waldvogel's case, the patients were elderly patients with arteriosclerosis. Rather large trocars were commonly used for thoracentesis. Puncture of the lung was quite accidental in most cases. All these cases were autopsied and although the patients suffered from severe diseases (pneumonia and tuberculosis), death was generally due to the puncture of an atheromatous vessel with hemorrhage into the pleural cavity, into the

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*Read (by invitation) before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery, at Atlanta, Ga., April 4, 5, and 6, 1938.

lung, and occasionally into the bronchi and trachea. The patients developed pulmonary edema and asphyxia. These observers warned against the unnecessary use of probatory puncture of the pleural cavity in elderly arteriosclerotic patients.

In 1909, Horder,[18] unaware of previous reports, described the procedure in *Lancet* as "Lung Puncture; A New Application of Clinical Pathology."

In the same year, Colby,[19] reporting in the *Journal of the Tennessee Medical Association,* mentioned twelve cases and reported several in detail, in which he performed puncture of the lung as a therapeutic measure in unresolved pneumonia. He obtained good results and no untoward complications.

Pierre Duval[20] in 1917, presented to the Société de Chirurgie de Paris, his experiences in the management of war injuries of the lungs. He mentioned cases of bullet extraction from the lung, in which forceps were inserted directly into lung tissue in order to grasp and remove the foreign body. Of 117 cases treated conservatively for bullet wounds of the lungs (no open operation) there were only eleven infections of the pleura and only one death occurred due to pleuropneumonitis.

In 1917, Hirschfeld[21] reported experimental work concerning the diagnosis of malignant tumors by probatory puncture and aspiration. He found the method satisfactory. Deelman,[22] in 1918, confirmed these observations and reported six cases of various malignant tumors in which the diagnosis was established by histologic examination of tissue fragments obtained by puncture with a needle. Hirschfeld and Deelman did not mention the aspiration of tumors of the lung.

Josefson,[23] in 1919, and Müller,[24] in 1927, described their experiences with puncture of tumors and aspiration by means of a needle. They made no specific mention of aspiration of tumors of the lung.

Ellis,[25] of the Memorial Hospital, in 1922, made an ante-mortem diagnosis of carcinoma of the lung from the preparation of material found clinging to an aspirating needle which had been used for a thoracentesis.

Martin and Ellis,[26] in 1930, reported sixty-five malignant neoplasms proved by needle puncture and aspiration. Two of the sixty-five neoplasms were primary carcinomas of the lung. Martin and Ellis, unaware of previous isolated reports, perfected the technique and proved the value of the diagnostic method. They are credited with definitely establishing the procedure in the field of neoplastic diseases, and with popularizing *aspiration biopsy.*

Stewart,[27] stimulated by the early work of Horder, reported from Edinburgh in 1930, the results of diagnostic puncture of the lung in seventeen cases of acute lobar pneumonia. There were no complications, and he concluded that the method was of value in cases of acute lobar pneumonia without sputum, in which culture and typing were desired.

Ferguson[28] reported the value of needle puncture and aspiration in the diagnosis of prostatic neoplasms. Coley, Sharp, and Ellis[29] reported the method in the diagnosis of bone tumors. Sharp,[30] in 1931, reported an additional case of cancer of the lung from the Memorial Hospital and reviewed the two previous cases (Martin and Ellis). Fernandez and Tobias,[31] in the same year, used the method successfully.

F. W. Stewart,[32] in 1933, reported the value of aspiration biopsy in general, on the basis of an experience in the study of smears of material obtained with an 18 gauge needle from some 2,500 tumors. He found aspiration biopsy a practical method of tumor diagnosis and stated, "Diagnosis by aspiration is as reliable as the combined intelligence of the clinician and pathologist makes it."

In 1934 Martin and Ellis[33] described the technique in detail. Martin and F. W. Stewart,[34] have recently summarized the advantages and limitations of aspiration biopsy. Pavlovsky,[35] of Buenos Aires, and more recently, Müller,[36] reporting in the year book of the Nederlandsch-Indisch Kankerinstituut, for 1937, found aspiration biopsy a reliable and expeditious method for the diagnosis of tumors.

Sappington and Favorite,[37] in 1936, in a review of "Lung Puncture in Lobar Pneumonia," mentioned twenty-three reports of various investigators and more than 2000 cases in which these various investigators performed puncture of the lung for bacteriologic purposes. In this large series there was but one death or serious complication (Thomas and Parker[38]). In this case the chest was being tapped for pleural fluid and the lung was accidentally punctured. After careful review of this subject, Sappington and Favorite conclude that puncture of the lung by a needle is a reasonably safe and useful diagnostic procedure. In addition to a review of the subject they report sixty cases of lobar pneumonia (fifty-seven pneumococcic and three streptococcic), in which the patients were subjected to diagnostic punctures of the lung. Positive cultures suitable for typing were obtained in 90 per cent, half of these within six hours. They advocate the method for early bacteriologic diagnosis and typing in the early case of pneumonia in which sputum is not available.

According to the authors, the incidence of empyema in the above mentioned fifty-seven cases of pneumococcus pneumonia was 5.3 per cent; this compares favorably with the incidence in Cecil's Bellevue Hospital series of 1,913 cases of all types, in which the incidence of empyema averaged 5.1 per cent. If the total number of punctures (including repeats) in the fifty-seven cases be used as a basis for the occurrence of empyema, the authors state that the incidence was but 4.6 per cent.

Lavalle,[39] professor of surgical pathology at the National University of Buenos Aires, has used puncture of the lung in the treatment of tuberculosis, and mentions over 100 cases of pulmonary tuberculosis which have received punctures of the initial nucleus in the lung as a

means of liberating autovaccines and providing specific antibodies against systemic tuberculosis. He regards the procedure of puncture of the lung as innocuous and states that it is performed regularly in the outpatient department.

Lanari and Pavlovsky,[40] in a recent report from Buenos Aires (1937), have analyzed their results in the diagnosis of cancer of the lung by needle puncture and aspiration. They obtained thirteen positive punctures of the lung (carcinoma) in twenty-four suspected cases. They conclude that in the presence of clinical and radiographic evidence of a neoplasm of the lung one should use the method without hesitation, particularly when bronchoscopy and sputum examinations have not established a definite diagnosis.

The purpose of this study is to report the results in ninety-two cases (suspected of being primary cancer of the lung), in which aspiration biopsies of the pulmonary lesions were performed in the Memorial Hospital during the eleven-year period, 1927 to 1937, inclusive.

By aspiration biopsy is meant the insertion of an aspirating needle directly into the tumor in order to obtain a plug of tissue for immediate smear and quick stain. (Martin and Ellis.[33]) This procedure has been used not only to prove the malignant nature of suspected primary lung tumors, but also to obtain definite histologic evidence in suspected metastasis to the lungs from a distant healed primary cancer.

One should not feel justified in aspirating a lesion of the lung, suspected of being a metastasis, until an exhaustive search has failed to locate a primary cancer elsewhere in the body. For example, complete studies of the kidneys including intravenous urography or retrograde pyelography may disclose evidence of silent primary cancer of the kidney. There seems but little reason for performing an aspiration biopsy of a suspected metastatic lesion in the lung, especially with a known active primary cancer elsewhere in the body. There may be some justification, however, for the early investigation of a solitary shadow in a lung field appearing a number of years after an apparently cured primary lesion. This report is primarily concerned with an evaluation of aspiration biopsy as an early effective diagnostic aid in suspected primary cancer of the lung.

Histologic evidence of cancer is of importance and a decided advantage in the proper management of a patient with a primary tumor of the lung. High voltage roentgen rays, radical surgery, or combinations including interstitial irradiation can be better evaluated if definite histologic proof of cancer exists.

Thoracic surgeons have reported their experiences in the surgical management of primary tumors of the lung. Many have expressed the need of some method of definitely proving preoperatively the diagnosis in the parenchymal and peripheral lesions. Such tumors located often in an upper or lower lobe, or far out in the parenchyma beyond a pri-

mary or secondary branch bronchus, are generally inaccessible for bronchoscopic biopsy. Aspiration biopsy is of particular value in this group of pulmonary lesions. These particular early tumors may be favorably located for lobectomy or pneumonectomy. Also, in this group, roentgen-ray therapy offers the possibility of a better depth-dose and a more accurate centering of the rays over the tumor mass. Hilar lesions arising in the bronchus or having eroded a bronchus are readily diagnosed by bronchoscopy. Such hilar lesions are often unfavorably located for surgery or roentgen-ray therapy.

Exploratory thoracotomy for inspection and palpation of the tumor has been mentioned as an aid in diagnosis where bronchoscopic methods have failed. Such a procedure naturally must depend upon the surgeon's clinical judgment based on past experience in the actual visualization and palpation of malignant tumors of the lung. Experience has shown that such clinical judgment is not always dependable and without error. Wedge or punch biopsy at the time of the exploratory thoracotomy has also been mentioned as a diagnostic procedure. Thoracotomy and thoracoscopy have been used in some clinics as diagnostic procedures and have been helpful in proving widespread pleural and regional metastasis. Thoracoscopy has saved patients needless exploratory operations.

Some surgeons have suggested that needle biopsy does not appear to be a safe procedure, that it may lead to infection, and that a negative report does not eliminate the possibility of a malignant tumor. Certainly the latter may be said to be true. However, in this series of ninety-two aspirations of the lung, none of the patients experienced undue complications. The patients exhibited no noticeable infections, no pleural shock or mediastinal shifting; no unusual pain, cough, or hemorrhage appeared as a complication in this series. Pain, cough, hemoptysis and partial collapse of the lung due to pneumothorax, occasionally occurred, but the symptoms were not severe and were readily controlled. The theoretical objection of an induced pleuro-pneumonitis or empyema due to a perforation of an infected bronchus or an infected tumor has not been observed in this series.*

Exploratory thoracotomy and thoracoscopy have a place in the diagnosis of certain tumors of the lung. This report is not to condemn such procedures but to report the results of another diagnostic method.

INDICATIONS FOR ASPIRATION BIOPSY OF THE LUNG

At the present time, it is not the practice to aspirate routinely all suspected tumors of the lungs. Aspiration biopsy is used as a method of choice in that group of cases in which the roentgenographic evidence indicates that the tumor is not accessible by bronchoscopy or in which the condition of the patient would seem to indicate an unfavorable reac-

*Recently, since this study was undertaken, a patient developed a moderate degree of cellulitis of the chest wall at the puncture site.

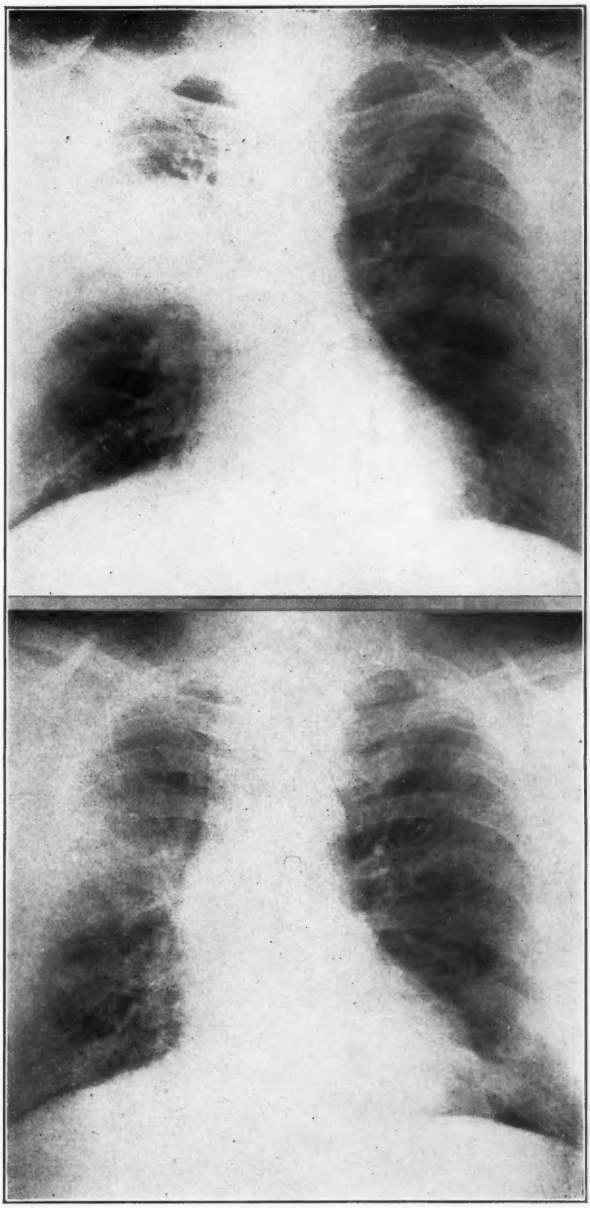
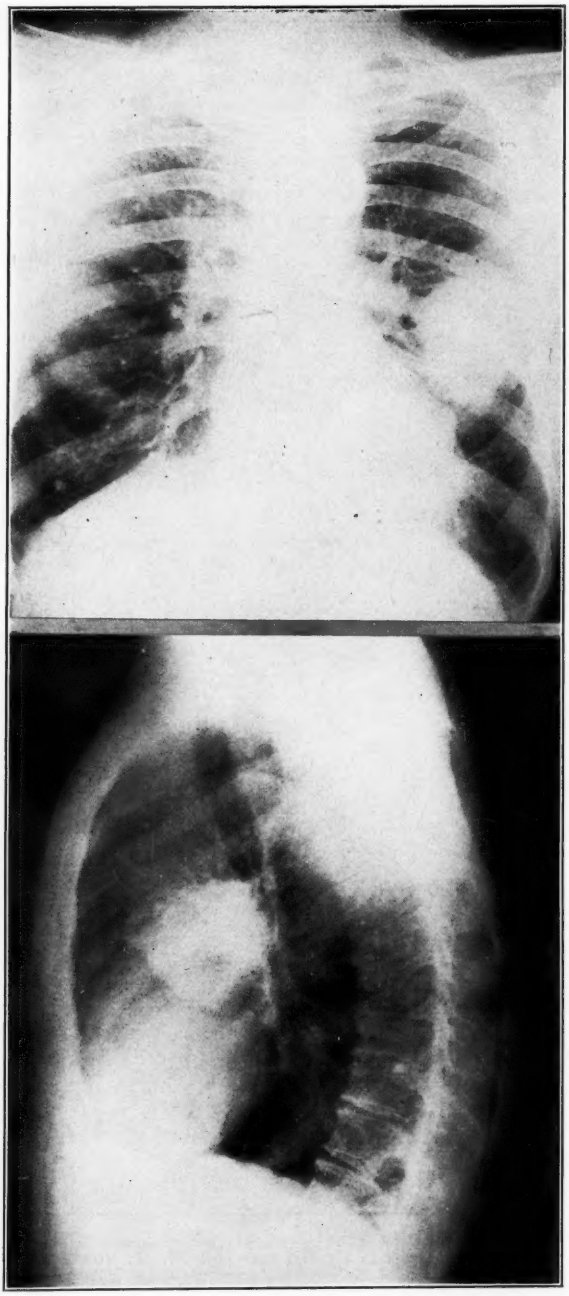


Fig. 1.—*A*, F. M., Dec. 29, 1936. Positive for carcinoma by aspiration biopsy. (Inoperable due to metastasis to cervical node.) *B*, F. M., Nov. 18, 1937, symptom-free following divided doses of high voltage roentgen rays. (Remains well to date.)



C.

D.

Fig. 1.—*C* and *D*, Bronchoscopy negative; proved to be carcinoma by aspiration biopsy.

tion to bronchoscopy (Fig. 1). Patients without accessible metastatic nodes for excision, or in whom previous repeated bronchoscopies have failed to obtain tumor tissue, are generally regarded as suitable for aspiration biopsy.

Regardless of the roentgenographic appearance of the lung fields, all the systems are carefully reviewed for a possible distant primary tumor, before subjecting the patient to an aspiration biopsy. If the roentgenograms present a circumscribed appearance suggestive of metastatic disease, the gastrointestinal and genitourinary systems are carefully checked by every means available. All questionable lung cases should have at least three specimens of sputum examined on separate occasions for the presence of specific organisms including the tubercle bacilli and for tumor cells. These studies should be routine prior to bronchoscopy or aspiration biopsy.

In this series care was used to avoid unnecessary aspirations and only thirteen patients received more than one attempted aspiration in order to obtain tissue. Repeated aspirations were not reported as failures where subsequent attempts produced a positive diagnosis.

TECHNIQUE

No casual attempt should be made to aspirate a tumor of the lung. The actual technique is simple, but considerable time and effort must be spent by the individual best suited to perform the procedure, in localizing and identifying the exact position of the tumor in the lung field. A working knowledge of roentgenoscopy is of considerable value in the localization of lung neoplasms. The exact site of the puncture can be outlined on the skin under roentgenoscopy. By the use of metal calipers the size and depth of the tumor can be determined, as well as the angle of inclination for the needle puncture (Fig. 2). The skin should be marked with the patient in the upright position; he may then be transferred to another room and aspirated with or without roentgenoscopic guidance (Fig. 3).

Roentgenoscopic guidance has been of value when technical difficulties were anticipated in aspirating small tumors less than 4 cm. in diameter. Though roentgenoscopic guidance is now being used a great deal at the Memorial Hospital, this added refinement is not regarded as necessary in the aspiration of the average tumor of the lung. Unless the operator is familiar with roentgenoscopy, the added technical difficulties may prove a disadvantage in maintaining a sterile field.

During the second half of 1937, roentgenoscopic guidance was used more often because of the smaller sized lesions encountered and for the purpose of evaluating this refinement in technique. For example, case J. M. (1937) presented a small left hilar mass, which on the lateral roentgenogram was noted to be extrinsic to the bronchus. The mass was localized under the roentgenoscope and found to be situated posterior

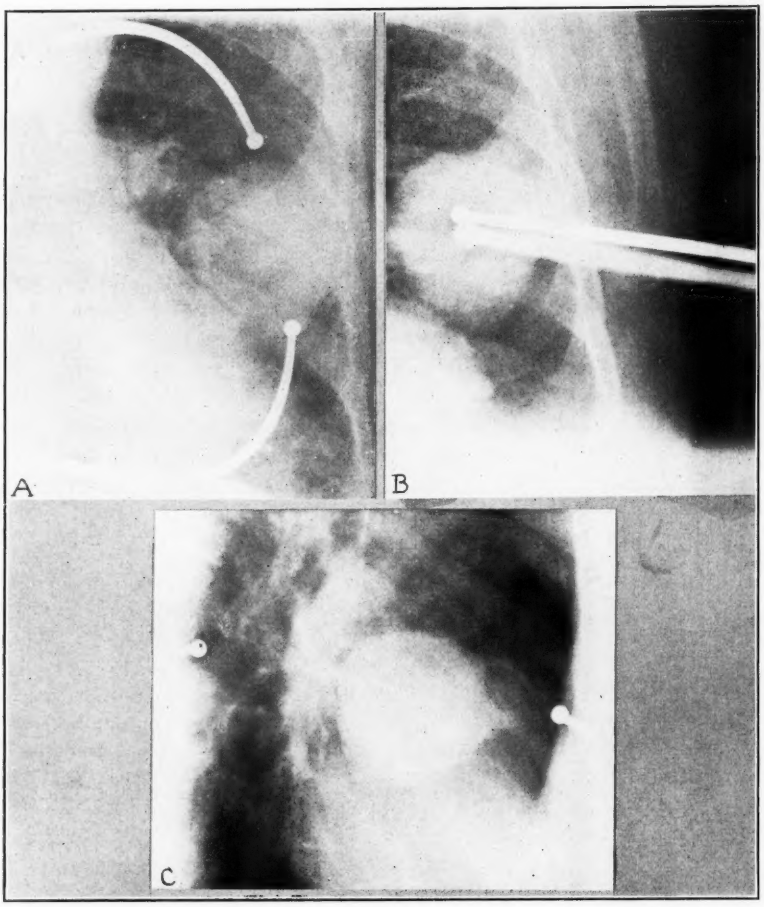
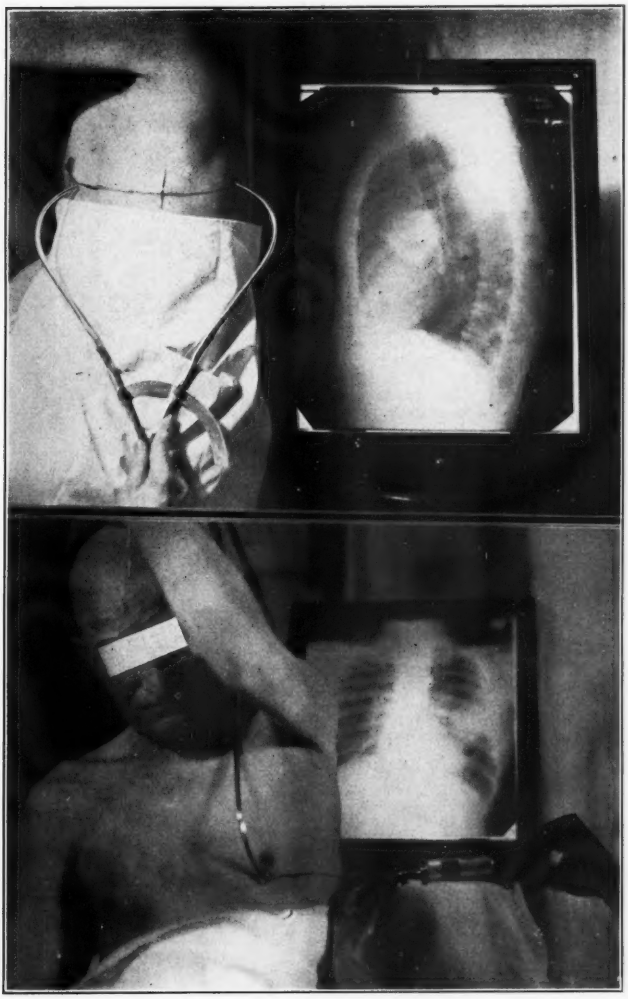


Fig. 2.—*A*, Anteroposterior (vertical plane); *B*, anteroposterior (horizontal plane); *C*, combined use of metal calipers, roentgenoscopy, and roentgenography in the localization and measurement of tumors of the lung.

to the heart and overlying the descending aorta. A member of the Fellowship staff, using roentgenoscopic guidance, successfully aspirated this tumor which measured approximately 4 by 5 cm. in diameter. The histology showed anaplastic carcinoma, presumably primary in origin.

If a roentgenoscope is not available, the roentgenograms, anteroposterior and lateral views, should be placed against the patient's chest while he is in the sitting position. (Method generally used at the Memorial Hospital up to 1936.) Bony landmarks, such as the clavicle, third interspace, fourth rib, etc., should be correlated with the roentgenograms. Measurements should be made and identifying marks mapped out on the skin of the patient in ink. The site of election for the in-

A.



B.

Fig. 3.—*A*, Reapplication of calipers following previous roentgenoscopic study and skin marking; *B*, aspiration biopsy by means of a 17 gauge needle and a twenty-cubic centimeter record syringe.

sertion of the aspirating needle should be marked with a cross mark and firmly visualized in case the iodine preparation of the field causes some fading of the skin markings.

It is important to realize the exact angulation and direction of the needle point. A knowledge of the estimated depth of the neoplasm is essential in order that the aspirator may anticipate the resistance of the

new growth as the needle point approaches the calculated depth. Such data can be fairly accurately obtained by careful measurements of the roentgenograms. Aspirating needles of 17 or 18 gauge caliber and measuring 10, 15, and 20 cm. in length, should be available.

A twenty-cubic centimeter record syringe, a sharp bistoury, and a two-cubic centimeter syringe with a skin needle and a longer needle for infiltrating the muscles, complete the sterile setup. A sterile test tube to receive blood or other fluid for examination, sterile sponges, rubber gloves, and at least four sterile glass slides complete the tray. If the slides are sterile, an initial failure to obtain and smear typical tissue will not cause a break in sterile technique. If there is much delay a new syringe will be required because of the tendency of the plunger to stick.

After the preliminary calculations have been made the patient is given a hypodermic injection of morphine sulfate, one-sixth grain. Thirty minutes later the patient is made comfortable in the sitting position on the examining table, with his feet over the edge and resting on a support. The procedure is carefully explained to the patient and he is reassured and requested to remain perfectly still, his hands on his knees. An assistant or nurse may steady the patient in front or from behind according to the site of election.

The skin is shaved and prepared with alcohol and iodine. The patient is requested to breathe in another direction. The patient's head is turned aside by the nurse. Drapes are not necessary. A skin wheal is made at the site of election and the deeper structures infiltrated with a few cubic centimeters of 1 per cent novocain. A puncture is made in the skin with the bistoury, and the aspirating needle (attached to the syringe) is slowly and carefully inserted into the lung in accordance with the calculations. Intermittent suction is applied as the needle penetrates the lung tissue. In the event that blood or air is encountered during the aspiration, due to the perforation of a vessel or a bronchus, the syringe should be disconnected, emptied, and the needle withdrawn slightly. The procedure is then continued by inserting the needle at a slightly different angle.

A sense of touch is soon developed which commonly gives an index to tumor tissue and lung tissue. If the patient coughs and complains of pain he is reassured and quieted. Such a reaction is to be expected and is more from fear and anticipation of severe pain than from actual discomfort. In no case in this series was it found necessary to discontinue an aspiration of a lung due to the patient's reaction.*

When tumor tissue is encountered, there will be as a rule noticeable increased resistance. This may be described as a "leathery feel." In

*A recent aspiration of the lung was associated with a collapse of the patient due to a partial stroke (probably air embolism) occurring at the end of the procedure. The patient was a 69-year-old male. The patient was hospitalized and recovered from his hemiplegia. A definite diagnosis of carcinoma of the lung was made from the aspirated material.

Fig. 4.—*A*, Aspirating needle within the tumor (suction maintained); *B*, material is transferred to a slide; *C*, material is firmly smeared; *D*, stained smear reveals carcinoma.

solid carcinoma considerable pressure is necessary in order to insert or withdraw the needle. If no such resistance is encountered, slight manipulation with withdrawal and reinsertion of the needle, enables the aspirator to locate the mass with the needle point. Constant suction should be applied and maintained as the needle enters the tumor.

Maintaining suction, the needle is inserted a centimeter or more into the area of resistance; the syringe and needle are than rotated (to loosen tissue), the needle withdrawn slightly and inserted further into the tumor in a different angle to complete a "*Y*." The needle and syringe are again rotated and suction is maintained as the needle is withdrawn from the mass. As the needle approaches the presumable limits of the tumor, suction is slowly released, the syringe disconnected, and the needle removed from the chest.

Frequently blood and frothy material are sucked into the syringe. This material should be removed at once and inspected. Particles of suspected tumor tissue should be smeared between slides. The blood clot should be saved for section. Choice material is often found in this manner rather than from the plug filling the end of the needle. The material within the needle should be removed either by the stylet or by blowing the tissue onto the slide by means of the syringe. The tissue should be flattened and smeared firmly between glass slides (Fig. 4). If tissue is not obtained, another aspiration can generally be accomplished before the termination of the sedation.

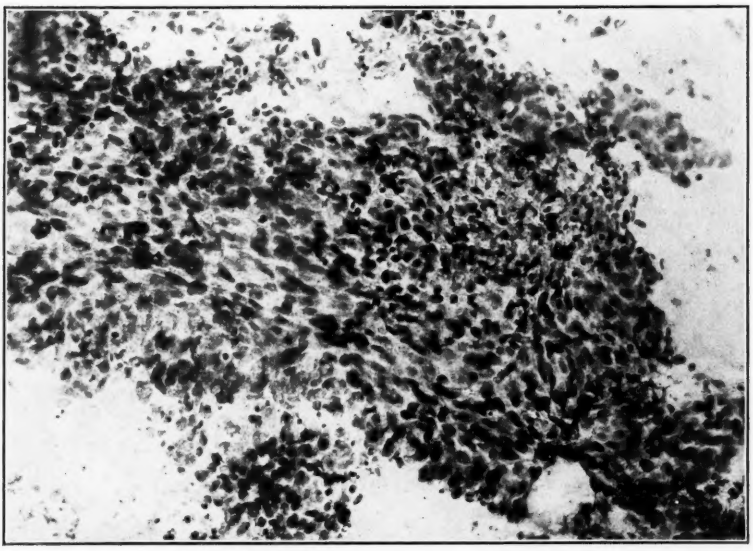


Fig. 5.—Case in which aspiration biopsy revealed carcinoma.

In the Memorial Hospital it is routine practice to send the smear immediately to the pathology laboratory where it is stained and interpreted. A report is generally available within ten minutes. During the interval the patient is kept in position and a new tray prepared in the event that a second aspiration is deemed advisable. Suspicious cells or lack of adequate material justifies a second attempt. Immediate

reaspiration generally overcomes previous faulty technique. Pathologists not familiar with interpreting aspiration smears may prefer to have the material carefully collected in one clump, fixed, and paraffin sections prepared. Additional particles of tumor tissue may be obtained by irrigating the syringe and needle with saline and carefully collecting the particles.

In the majority, the patients in this series were aspirated in the outpatient clinic. They returned to their homes within an hour after the aspiration, by taxi or by subway. Some patients cough and complain of pain, others are indifferent to the procedure. Occasionally a patient will expectorate a slight amount of blood, particularly if a vessel or a bronchus is punctured. Certain patients need a prescription of codeine sulfate gr. 1, p.r.n. for the first night; however, most patients experience but little distress and are advised to take an aspirin tablet at bedtime. Added experience may increase our knowledge of the hazards not yet encountered from aspiration of tumors of the lung.

Though hospitalization is preferable, experience indicates that with due regard to the patient's general physical condition, aspiration biopsy of selected tumors of the lung may be regarded as a reasonably safe outpatient procedure, provided the patient is accompanied by a relative and is under the supervision of a family physician. The procedure is often less arduous for the patient than bronchoscopy.

In this institution a shortage of bed space does not permit hospitalization for many of the common diagnostic procedures such as bronchoscopy, esophagoscopy, and gastroscopy. Such procedures are generally performed in special clinics with the aid of sedatives and topical anesthesia. The clinic patients, as a rule, return to their homes following such instrumentation.

INTERPRETATION OF RESULTS

The rise for the year 1937 (Fig. 6) does not represent results obtained as a method of choice but rather represents the coincidence of unfavorable cases for bronchoscopy, and bronchoscopic negatives or nontechnical failures. These cases may possibly represent earlier lesions, the result of recent cancer propaganda. This is not an attempt to discredit bronchoscopic biopsy, but an attempt to show the value of aspiration biopsy in establishing a diagnosis. Without aspiration biopsy one would be forced to resort to thoracoscopy or exploratory thoracotomy in order to obtain a biopsy in the inaccessible or peripheral lesions (Table I). Even in more centrally located tumors, months may pass before the tumor erodes a bronchus sufficiently to enable one to obtain tissue through the bronchoscope for definite proof of cancer.

If the figures and percentages (Fig. 7) are to be regarded as sufficiently representative of the distribution and character of carcinoma of the lung in general (which of course is doubtful), it may be assumed

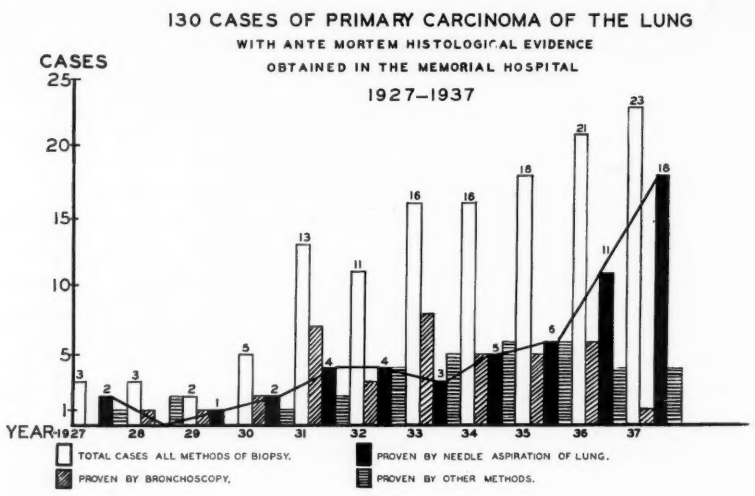

130 CASES OF PRIMARY CARCINOMA OF THE LUNG
WITH ANTE MORTEM HISTOLOGICAL EVIDENCE
OBTAINED IN THE MEMORIAL HOSPITAL
1927–1937
CASES
YEAR 1927 28 29 30 31 32 33 34 35 36 37
TOTAL CASES ALL METHODS OF BIOPSY.
PROVEN BY BRONCHOSCOPY.
PROVEN BY NEEDLE ASPIRATION OF LUNG.
PROVEN BY OTHER METHODS.

Fig. 6.

that one-third and more of neoplasms of the lung, if observed during the early stages, will not be accessible for bronchoscopic biopsy. Subsequent growth of the tumor often does permit a bronchoscopic diagnosis. However, such delay seems unwarranted. Results indicate that the majority of these cases can be proved by needle puncture and aspiration.

An analysis of Fig. 6 would seem to indicate a rise in the incidence of cancer of the lung. One must assume however that this rise in such

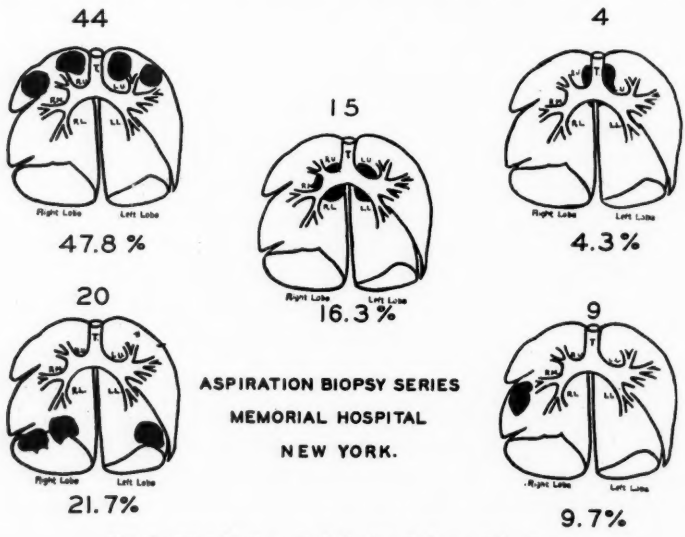


Fig. 7.—Distribution of ninety-two lesions of the lung.

TABLE I

CANCER OF THE LUNG, ASPIRATION BIOPSY SERIES*

(Memorial Hospital)

	PROVED CASES	PER CENT
Aspiration biopsy without preliminary bronchoscopy	24	42.8
Bronchoscopic failures proved by aspiration biopsy	32	57.1
Total	56	

*Age distribution:
Median age (for 56 cases), 53 years.
Age of youngest patient, 33 years.
Age of oldest patient, 73 years.

a small series indicates a more widespread recognition of the cardinal symptoms referable to neoplasms of the lung.

Starting in 1929, the number of cases proved by aspiration biopsy represent roughly one-third and more of all cases biopsied in this clinic. Of 130 proved cases of carcinoma of the lung from 1927 to 1937, inclusive, 56, or 43 per cent, were proved by needle puncture and aspiration. Table II does not represent all the cases treated during that interval but merely those cases in which microscopic proof was obtained in this clinic. Approximately two-thirds of the cases treated for cancer of the lung are referred to the Memorial Hospital with a positive biopsy (bronchoscopic) obtained elsewhere.

TABLE II

PRIMARY CANCER OF THE LUNG WITH ANTE-MORTEM HISTOLOGIC EVIDENCE

(BIOPSIED IN THE MEMORIAL HOSPITAL, 1927 TO 1937)

	CASES	PER CENT
Bronchoscopy	39	30
Aspiration biopsy (needle puncture)	56	43
Excision of metastatic node or nodule	14	10.7
Aspiration of metastatic node	13	10
Miscellaneous:	8	6.1
Pleural Fluid (4)		
Thoracotomy (3)		
Aspiration of Ileum (1)		
Total	130	

The atelectasis and infection secondary to primary cancer of the lung, so commonly described, were not noticeable features in the series of aspirated cases. It is in the group that is referred, biopsied elsewhere and proved by bronchoscopy, that atelectasis and infection have occurred, or have existed previously. The clinical course of most of this group shows bulky hilar involvement, rapid extension, and terminal disease. Certainly many of these cases fall into a hopeless group even from the beginning of their initial symptoms. Though no definite conclusions can be made at this time, the type of case now being referred to the Memorial Hospital for diagnosis, suggests the rather frequent occurrence of *silent,* early primary carcinoma of the lung. Such tumors arise outside

the main bronchial tree, are often discovered accidentally, and are not accessible to bronchoscopic biopsy until a rather late stage of the disease.

Fig. 8 shows the relative increase in the percentage of positive aspirations. Much of this increase has been due to the type of lesion encountered. Closer attention to localization of the lesion, and the application of previously mentioned technical details, account for the relative increase in successful aspirations since 1934. Roentgenoscopic guidance has been of value in the 1937 group of cases.

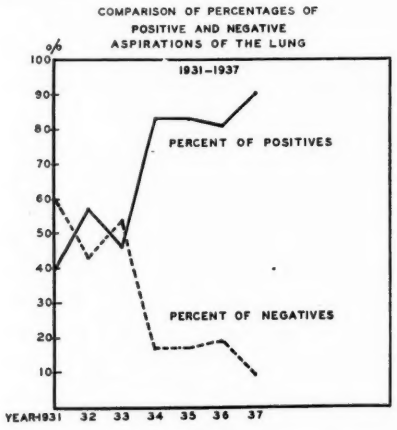


Fig. 8.

Proved failures mentioned in Table III are regarded as those cases in which aspiration biopsy was attempted and in which definite proof was finally obtained by other methods. These failures are reported in further detail. A negative aspiration is not accepted as proof against cancer or in favor of a benign lesion. Subsequent bronchoscopy, re-aspiration, or other methods may be used in accordance with the clinical picture.

Significant negative aspirations are those failures in which no definite proof of cancer was ever obtained other than the clinical and roentgenographic evidence. Most of these patients received repeated bronchoscopic biopsies, and definite tumor tissue was never obtained. Table IV indicates that one of this group was autopsied and no cancer found, the lesion being inflammatory. Five of the patients are alive and clinically well with no evidence of cancer. Another patient subsequently developed clinical Hodgkin's disease. It must be said that in most of the twelve remaining questionable cases there was certainly clinical evidence of carcinoma of the lung during the patients' stay in this clinic. Some died of symptoms of cerebral metastasis in other institutions. Undoubtedly most of this latter group of undiagnosed patients would have presented some source of biopsy material had they remained under the guidance of this clinic or returned for terminal care. In this group, thoracoscopy or ex-

TABLE III

ASPIRATION BIOPSY OF NINETY-TWO TUMORS OF THE LUNG

(MEMORIAL HOSPITAL, 1927 TO 1937)

POSITIVE ASPIRATIONS			NEGATIVE ASPIRATIONS		
	CASES	PER CENT		CASES	PER CENT
Carcinoma	56	60.8	Proved failures	6	6.5
Malignant tumor	4	4.3			
Benign lesions	7	7.6	Significant negatives	19	20.6
Total	67	72.7	Total	25	27.1

TABLE IV

ANALYSIS OF NEGATIVE ASPIRATIONS				NO. OF CASES
Proved carcinoma of the lung				6
Significant negatives				19
Proved inflammatory (autopsy—no cancer)	1	Clinical cancer (now dead)	7	
Alive and well (no clinical cancer)	5	Clinical cancer (now alive)	1	
Clinical Hodgkin's disease (alive)	1	Clinical Cancer (lost—transfer)	4	
Total				25

TABLE V

BENIGN LESIONS OF THE LUNG PROVED BY ASPIRATION BIOPSY

Dermoid cyst	1	Solitary cyst of lung	1
Echinococcus cyst	1	Bilateral apical cysts	1
Lung abscess	2	Encapsulated empyema (and lung abscess)	1
Total			7

ploratory thoracotomy might have established a diagnosis in selected cases. Either the patients presumably did not have cancer, or the lesions, if cancer, were so situated and of such character that biopsy by other methods was unsuccessful or impracticable. A negative aspiration biopsy in this group is regarded as significant, though naturally not as proof against cancer.

In the positive cases (Table III) the diagnosis was based on the stained smears, such as shown in the microphotographs (Fig. 5). The decision regarding primary or metastatic carcinoma must be based on the clinical evidence. The appearance of the stained smear is of value in correlating clinical evidence in establishing a diagnosis of primary carcinoma of the lung.

The diagnosis of the dermoid cyst (Table V) was established on the fluid which contained cholesterin crystals and other particles suggestive of a dermoid. The congenital cysts yielded fluid characteristic of benign cysts and there was no evidence of tumor cells. The echinococcus cyst yielded daughter cysts and the lung abscesses were proved by typical pus and bits of lung tissue free of tumor cells.

TABLE VI

ASPIRATION BIOPSY OF NINETY-TWO TUMORS OF THE LUNG

(Memorial Hospital, 1927 to 1937)

YEAR	CASE	AGE	SEX	CLINICAL DIAGNOSIS	ROENTGENOGRAPHIC DIAGNOSIS	BRONCHOSCOPIC					BIOPSY ASPIRATION				MISCELLANEOUS	HISTOLOGIC DIAGNOSIS
1927	A.S.	67	M	Endothelioma pl.	Mal. tumor left apex.						+	+				Carcinoma
	C.L.	48	M	Mediast. tumor	Car. rt. lung and mediastinum						+					Carcinoma
1928	G.P.	47	F	Car. rt. upper lobe	Tumor rt. upper lobe	−					−					No tumor cells
1929	N.S.	45	M	Car. rt. upper lobe	Tumor rt. upper lobe	−					+					Carcinoma
1930	D.C.	56	F	?Dermoid cyst	Cyst, lt. upper lobe						+					Dermoid cyst
	J.Z.	53	M	Car. rt. lower lobe	Car. rt. lower lobe	−					+					Carcinoma
	S.C.	54	M	Car. lt. lower lobe	Car. lt. lower lobe	−					+					Carcinoma
1931	*T.F.	60	M	Car. lt. bronchus	Car. lt. bronchus	−	−				−	−	−			No tumor cells
	M.A.	64	F	Car. lt. lower lobe	Car. lt. lower lobe	−					-					Fibrin only
	J.B.	57	M	Car. lt. lower lobe	Car. lt. lower lobe		+				+					Carcinoma
	S.G.	55	F	Car. rt. lower lobe	Car. rt. lower lobe						+					Carcinoma
	G.H.	68	M	Car. rt. middle lobe	Car. rt. middle lobe	−	−				−					No tumor cells
	*M.K.	60	F	Car. rt. lower lobe	Car. rt. lower lobe		+				-					Carcinoma
	L.M.	56	M	Car. rt. lower lobe	Car. rt. lower lobe						-					No tumor cells
	J.R.	41	M	Car. lt. upper lobe	Car. lt. upper lobe	−					+					Carcinoma
	S.S.	44	M	Car. rt. middle lobe	Car. rt. middle lobe	−	−	−			+	-	+			Carcinoma
	A.D.	63	M	Car. lt. upper lobe	Car. lt. upper lobe	−	(elsewhere)				−					No tumor cells

*Subsequent proof of cancer obtained by other methods.

TABLE VI—(CONT'D)

1932	C.S.	37	F	Mediast. tumor	Tumor lt. upper lobe	–					–					Mucin only
	R.F.	45	M	Car. lt. hilum	Car. lt. bronchus	–					–					No tumor cells
	A.L.	60	M	Car. rt. upper lobe	Car. rt. upper lobe						+					Carcinoma
	C.M.	56	M	Car. lt. upper lobe	Car. lt. upper lobe		–				–	+				Carcinoma
	*H.S.	56	M	Car. lt. upper lobe	Car. lt. upper lobe						–	–			(Excised node+) for carcinoma	No tumor cells
	D.W.	50	M	Car. rt. upper lobe	Car. rt. upper lobe	–	–	–	–					+		Carcinoma
	H.N.	33	M	Mediast. tumor	Mediast. tumor	–					+					Carcinoma
1933	R.C.	46	M	Lung abscess	Infilt. rt. apex.						+				(Autopsy+, carcinoma)	Malignant tumor
	*J.C.	51	M	Car. lt. upper lobe	Car. lt. upper lobe	..					–				(Excised node+) for carcinoma	No tumor cells
	D.C.	67	M	Car. rt. lower lobe	Car. rt.—effusion								+			Carcinoma
	H.F.	53	M	Car. rt. lung?	Car. rt. bronchus	–	–				–					No tumor cells
	S.K.	58	M	Car. lt. lung?	Car. lt. hilum	–					–					No tumor cells
	Y.L.	46	F	Car. lt. upper lobe	Car. lt. upper lobe						+					Carcinoma
	*M.W.	52	M	Car. rt. upper lobe	Car. rt. apex	–					–				(Autopsy+, carcinoma)	15 c.c. fluid
	C.N.	36	M	Car. rt. upper lobe	Car. rt. upper lobe						–	–				No tumor cells
	V.C.	26	M	Tumor rt. middle lobe	?Car. rt. middle lobe	–					+					Echinococcus cyst
	P.E.	57	M	Mediast. tumor	Cyst, or tumor lt. hilum						–	+				Malignant tumor
	M.L.	39	F	Tumor lt. upper	Car. lt. upper lobe	–	(elsewhere)				–					No (?) tumor cells
	C.M.	57	M	Tbc. or Car.	Lung absc. lt. hilum	–	–				–					No tumor cells
	S.M.	44	M	Car. lt. upper lobe	Car. lt. upper lobe						+					Carcinoma
1934	E.B.	52	M	Car. rt. upper lobe	Car. or cyst. rt. apex						+					Carcinoma
	J.B.	56	M	Car. rt. upper lobe	Car. rt. upper lobe	–					+					Carcinoma
	J.G.	65	M	Car. rt. lower lobe	Car. rt. lower lobe						–					20 c.c. fluid
	W.G.	56	M	Car. rt. upper lobe	Car. rt. upper lobe	–					+					Carcinoma
	F.H.	50	M	Tumor lt. upper lobe	Car. lt. upper lobe						+					Carcinoma
	J.Y.	72	M	Car. lt. hilum	Car. lt. hilum						+	+				Carcinoma

TABLE VI—(CONT'D)

YEAR	CASE	AGE	SEX	CLINICAL DIAGNOSIS	ROENTGENOGRAPHIC DIAGNOSIS	BRONCHOSCOPIC					BIOPSY ASPIRATION				MISCELLANEOUS	HISTOLOGIC DIAGNOSIS
1935	J.C.	62	M	Tumor rt. lung	Tumor rt. hilum	−					−	+				Carcinoma
	J.F.	56	F	Car. lt. upper lobe	Car. lt. upper lobe						+					Carcinoma
	J.K.	48	M	Car. rt. bronchus	Car. rt. bronchus	−					−	−	+			Carcinoma
	J.M.	37	M	Tumor rt. upper lobe	Car. rt. upper lobe	−					+					Carcinoma
	T.M.	39	M	Car. rt. middle lobe	Car. rt. middle lobe						+					Malignant tumor
	*F.R.	52	M	Car. rt. lung	Car. rt. hilum	−	+				−	−				Carcinoma
	M.S.	36	M	Tumor rt. lung	?Sa. rt. lower lobe						+					Carcinoma
	L.T.	56	M	Car. rt. upper lobe	Car. rt. upper lobe		+				+					Carcinoma
	H.W.	68	M	Mediast. tumor	Mediast. tumor						−	+				Malignant tumor
	G.M.	45	M	Car. rt. middle lobe	Rt. lung abscess						+					Pus, no tumor cells
	M.G.	47	M	Car. rt. middle lobe	Meta. car. rt. lung	−	−				−	−				No tumor cells
	F.R.	35	M	Car. rt. lower lobe	Cyst. rt. lower						+					60 c.c. fluid
1936	M.C.	50	M	Car. lt. hilum	Car. left hilum						+					Carcinoma
	N.B.	49	M	Car. lt. upper lobe	Car. lt. upper lobe	−					+					Carcinoma
	J.B.	39	M	Car. lt. upper lobe	Car. lt. upper lobe	−					+					Carcinoma
	J.L.	55	M	Car. lt. upper lobe	Car. lt. upper lobe						+					Carcinoma
	L.M.	55	M	Car. lt. lower lobe	Car. lt. hilum	−					−					(?) cells
	S.S.	67	M	Car. lt. lower lobe	Car. lt. lower lobe						+					Carcinoma
	C.S.	61	M	Car. rt. upper lobe	Car. rt. upper lobe	−					+					Carcinoma
	A.S.	60	M	Car. rt. upper lobe	Car. rt. upper lobe	−					−	+				Carcinoma
	L.W.	43	M	Car. rt. upper lobe	Car. rt. upper lobe	−					+					Carcinoma
	L.S.	54	M	Encap. emp. ?car.	Encapsulated fluid, left base	−					+					Pus, no tumor cells
	V.B.	48	M	?Car. rt. middle lobe	Tumor rt. middle lobe	−	−				+	+				Carcinoma
	B.B.	50	F	?Neoplasm lf. apex	?Tbc. lt. apex						−					No tumor cells
	F.M.	54	M	Car. rt. middle lobe	Car. rt. middle	−					+					Carcinoma
	A.C.	42	F	?Car. lt. lower lobe	?Mass lt. lower	−					−					No tumor cells
	M.B.	57	M	Mediast. tumor	Bilat. apical cysts	−					+	+	+			60 c.c. fluid
	J.R.	41	M	Mediast. tumor	Substernal thy.						+					No tumor cells Carcinoma

TABLE VI—(CONT'D)

1937	J.B.	58	M	Car. rt. upper lobe	Car. rt. upper lobe	-	(elsewhere)			+					Carcinoma
	F.D.	45	M	Car. rt. lung	Car. rt. upper lobe	-				+					Carcinoma
	H.G.	53	M	Car. rt. base	Car. rt. lower lobe	-				+	+				Carcinoma
	S.K.	62	M	Car. rt. upper lobe	Car. rt. upper lobe					+					Carcinoma
	I.M.	47	M	Car. rt. lung	Infiltration rt. lung base	-				+					Carcinoma
	H.B.	65	M	?Car. rt. upper lobe	Inflam. rt. apex					+				(Surgery+, for abscess)	Pus, no tumor cells
	R.G.	62	M	?Tbc., ?car.	Infilt. rt. base					-					Liver cells
	A.J.	43	M	Car. lt. bronchus	Car. lt. hilum		-			-					Lymphoid cells
	H.C.	51	M	Car. lt. upper lobe	Car. lt. upper lobe	-				+					Carcinoma
	A.F.	57	M	Car. lt. hilum	Car. lt. hilum	-				+					Carcinoma
	H.F.	73	M	Car. lt. upper lobe	Car. lt. upper lobe	-				+	-				Carcinoma
	A.F.	68	M	Car. rt. upper lobe	Car. rt. upper lobe	-				+					Carcinoma
	A.L.	49	M	Car. rt. upper lobe	Car. rt. apex					+					Carcinoma
	J.M.	55	M	Car. rt. middle lobe	Car. rt. middle lobe	-				+					Carcinoma
	A.P.	63	M	Car. lt. lung	Car. lt. base	-				+					Carcinoma
	G.C.	69	M	Car. rt. lower lobe	Car. rt. base	-				-	+				Carcinoma
	J.S.	65	M	Car. lt. lower lobe	Car. lt. base					+					Carcinoma
	J.T.	39	M	?Tbc. rt. upper lobe	Car. rt. apex					-	+				Carcinoma
	J.M.	50	M	Mediast. tumor	Tumor lt. hilum					+					Carcinoma
	C.T.	40	M	?Aneurysm	Car. rt. upper lobe	-				+					Carcinoma
	E.P.	53	M	Car. rt. upper lobe	Car. rt. upper lobe					+					Carcinoma

TABLE VII

TWENTY-FIVE NEGATIVE ASPIRATION BIOPSIES OF TUMORS OF THE LUNG

(Memorial Hospital)

YEAR	CASE	LOCATION OF LESION	BRONCHOSCOPY				ASPIRATION BIOPSY				COMMENTS	END RESULT—1937
1928	G.P.	Right upper lobe	–				–				Clinically well, no evidence of cancer	Alive
1931	A.D.	Left upper lobe	–				–				Clinically cancer, no histologic proof	Died 3/4/32
	L.M.	Right lower lobe					–				? Cancer, no histologic proof	Refused therapy
	*M.K.	Right lower lobe		+			–				Primary cancer by bronchoscopy	Dead
	G.H.	Right middle lobe	–	–			–				No cancer at autopsy (inflammatory)	Dead
	M.A.	Left lower lobe	–				–				Clinically cancer, no histologic proof	Died 9/8/31
	*T.F.	Left hilar region	–	–			–	–	–		Cancer left upper lobe at autopsy	Dead (1933)
1932	*H.S.	Left upper lobe					–				Excised metastatic node showed cancer	Dead
	R.F.	Left hilar region	–				–				Clinically cancer, no histologic proof	Died 5/9/32
	C.S.	Left upper lobe	–				–				No histologic proof, (?) neurofibroma	Alive 9/20/37
1933	C.M.	Left hilar region	–	–			–				Clinically cancer, no histologic proof	Died 7/20/33
	M.L.	Left upper lobe	–	(elsewhere)			–				Clinically cancer, no histologic proof	Lost 11/14/34
	C.N.	Right upper lobe					–	–			Clinically well, no evidence of cancer	Alive
	*M.W.	Right upper lobe	–				–				Primary carcinoma of the bronchus—autopsy	Dead
	S.K.	Left hilar region	–				–				Clinically cancer, no histologic proof	Died 9/25/33
	H.F.	Right hilar region	–	–			–				Clinically cancer, no histologic proof	Died 9/28/33
	*J.C.	Left upper lobe	–				–				Excised metastatic node showed cancer	Dead
1934	J.G.	Right lower lobe					–				Clinically cancer, no histologic proof	Died 6/2/35
1935	M.G.	Right hilar region	–	–			–	–			Clinically well, no evidence of cancer	Alive
	*F.R.	Right hilar region			+		–	–			Carcinoma by subsequent bronchoscopy	Dead
1936	L.M.	Left hilar region	–				–				(?) Cancer, no histologic proof	Lost 6/23/37
	A.C.	Left lower lobe	–				–				Mass has regressed, appears benign	Alive
	B.B.	Left upper lobe					–				Recent roentgenograms appear negative	Alive
1937	R.G.	Right lower lobe					–				Aspiration showed liver cells, no proof	Lost 8/6/37
	A.J.	Left hilar region	–				–				Aspiration showed lymphoid tissue, (?) Hodgkin's disease	Alive

*Histologic proof of carcinoma obtained by other methods.

AN ANALYSIS OF THE PROVED TECHNICAL FAILURES

In these ninety-two cases of tumors of the lung aspirations were done by seventeen different physicians connected with the staff of the Memorial Hospital. In no case was a failure due to the incorrect interpretation of aspirated material by the pathologist.

Of the six proved technical failures (a) two were obtained by a physician who performed thirty or more of the aspirations; (b) two were obtained by two physicians who had no actual previous experience and were using the technique for the first time; (c) one was obtained by a physician who performed five of the aspirations; (d) one was obtained by a physician who performed three of the aspirations.

The following résumé indicates that the failure in each case was due to the failure to obtain tissue.

Failures:

1. No cells obtained on stained smear. A bronchoscopic biopsy obtained one month later was positive for anaplastic carcinoma.

2. No cells obtained on stained smear. An excised supraclavicular node which appeared at a subsequent date was positive for carcinoma; primary undetermined from section. (No bronchoscopy.)

3. No cells obtained on stained smear. A subsequent bronchoscopy was negative. An excisional biopsy of a femoral node showed "metastatic embryonal bronchogenic carcinoma, grade 4."

4. No cells obtained on stained smear. Bronchoscopy twelve days later was negative. Another aspiration biopsy was not productive of tissue. A bronchoscopic examination repeated one month later was successful and the specimen proved to be carcinoma.

5. No cells obtained on stained smear. A cyst containing 15 c.c. of fluid was encountered within the tumor. Fluid was negative for tumor cells. A subsequent bronchoscopy was negative. A thoracentesis during the terminal stages showed suspicious cells in the fluid. Autopsy disclosed a "primary mucous gland, adenocarcinoma of the bronchus."

6. No cells obtained on stained smear. There were two negative bronchoscopies. Autopsy two years later revealed "carcinoma of the left upper lobe."

The failures to obtain tissue in these cases may be explained by various reasons, such as deeply situated tumors or an overcautious attitude and a disinclination to repeat the procedure following failure to obtain tissue. As has been mentioned by Martin and Ellis,[33] the greatest single cause of failure to obtain tissue is the failure to maintain adequate suction during the manipulation of the aspirating needle after it is inserted into the tumor. This error probably accounted for failure in at least three of the six cases. In one case, fluid was obtained from a cyst within the tumor and no further attempt was made to aspirate solid material. The remaining failures were probably due to failure to

locate the tumor mass with the point of the aspirating needle. Roentgenoscopic guidance might have prevented these failures.

Of the thirteen patients that received more than a single puncture of the lung, nine on reaspiration yielded positive results. Of the four remaining negatives, two were subsequently proved, the one by autopsy, the other by bronchoscopy at a later date (included in failures). The two remaining reaspirated patients were reported negative. One is clinically well three years with no evidence of cancer. The other patient is without symptoms and without definite histologic proof of cancer.

SUMMARY

(1) Aspiration biopsy (needle puncture) of 92 different pulmonary lesions produced material sufficient to establish a positive diagnosis of carcinoma in 56 patients or 60.8 per cent of the cases.

(2) Of the 67 cases with positive aspirations, 56 were carcinoma, 4 were malignant tumors (not definitely classifiable) and 7 were benign lesions.

(3) In 19 of 25 cases in which no tumor cells were obtained by aspiration, either the subsequent clinical course of the patient, the failure of repeated bronchoscopic studies, or the disappearance of roentgenographic evidence, made the presumptive diagnosis of carcinoma of the lung doubtful or at least unconfirmed by histological evidence. (One case had no cancer at autopsy; 5 patients are living and clinically well; one patient is thought to have Hodgkin's disease; 12 presumably had cancer of the lung.)

(4) Six proved failures by aspiration biopsy are reported.

(5) Thirty-two cases of primary carcinoma of the lung, or 57.1 per cent of the 56 cases of cancer of the lung, were proved by aspiration biopsy following one or more previous bronchoscopic failures.

(6) Of the 92 cases, 54 received initial bronchoscopic studies. Attempted bronchoscopic biopsy resulted in no definite proof in 52 of 54 cases or 96.2 per cent nontechnical failures. Positive bronchoscopies were obtained in 2 of the 54 cases or 3.7 per cent.

(7) No significant complications occurred, attributable to aspiration biopsy, in this series of cases except one case of temporary hemiplegia.

(8) Of 130 cases of clinical primary carcinoma of the lung, with ante-mortem histologic evidence of cancer of the lung, biopsied (all methods) in the Memorial Hospital from 1927 to 1937 inclusive, 56 cases or 43 per cent were proved by aspiration biopsy. For the year 1937, 18 of 23 cases or 78.2 per cent were proved by needle puncture and aspiration.

(9) A high proportion of the cases of cancer of the lung referred to the Memorial Hospital for diagnosis present roentgenographic evidence of peripheral lesions inaccessible to bronchoscopy. Of the 92 cases, 16.3

per cent were hilar lesions, 83.5 per cent were peripheral and mediastinal lesions (47.8 per cent upper lobes, 21.7 per cent lower lobes, 9.7 per cent right middle lobes, and 43 per cent mediastinal).

CONCLUSIONS

(1) Aspiration biopsy of selected tumors of the lung is a valuable and relatively safe diagnostic procedure.

(2) Pathologists familiar with the appearance of the stained smears can make a diagnosis if tumor tissue is obtained.

(3) There is a substantial percentage of early primary cancer of the lung in which bronchoscopy will fail as a diagnostic procedure.

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DISCUSSION

DR. DAN C. ELKIN, ATLANTA, GA.—I believe most patients with carcinoma of the lung will not be operated upon unless a definite clean-cut diagnosis can be made. As Dr. Binkley has shown, this cannot always be done by removal of a piece of tissue by bronchoscopy; therefore, aspiration biopsy offers the best means of a definite diagnosis.

Likewise, there are many patients and patients' families who believe that tuberculosis is the cause of their cough and refuse operation or other treatment on that account. It is a case in question of this type that I would like to show. This man, in 1934, because of hemoptysis and chronic cough, had a diagnosis of tuberculosis, not unreasonable from the view of the apices of the lung. In 1936 he had treatment by pneumothorax, supposedly for tuberculosis, and was seen by me at this time. The fourth rib showed erosion, the aspiration biopsy, definitely carcinoma, and the diagnosis was established as inoperable. The patient is being treated by radiation.

DR. J. J. SINGER, LOS ANGELES, CALIF.—I think this procedure is very timely, particularly when the question of operability of carcinoma of the lung is definitely proved and the question now is how early can one find the lesion.

We have had a fair amount of experience with the lung puncture but it was always done when nothing else was found positive. The first picture that was shown with a lesion in the right apex was the one particularly difficult to diagnose by the bronchoscope or other methods. Failures are not uncommon, as demonstrated in a case recently in which two lung punctures were made which were negative; the surgeon operated the patient and took out a piece of the lung, apparently a piece of the tumor, and on section the report showed as only inflammatory tissue.

The question of puncturing the chest wall naturally does give one a little fear because if the pleural space is free (and sometimes one is surprised how often it is) one may get a pneumothorax and possible empyema. Embolism occasionally occurs.

One of the methods that was not described in the paper is a puncture of the chest wall after diagnostic pneumothorax has been done.

I have designed a small thoracoscope in which I use a small flat cannula with a flat knife which fits into the tube. By removing the knife one inserts a baby cystoscope; in the hub of this instrument there is a small opening through which one can insert a flexible biopsy forceps, and under direct vision one can see the undersurface of the lung and the pleura, and easily snip off a piece of tissue. If one wants to use a cautery or even a high frequency current he merely puts the appropriate instrument in this opening. That has helped us in many cases when biopsy through a puncture, or even aspiration of fluid, has failed.

DR. JEROME R. HEAD, CHICAGO, ILL.—I think Dr. Binkley is to be commended for emphasizing this very important and very often neglected method of diagnosis; as Dr. Elkin has said, the necessity of making an accurate anatomical diagnosis, in view of the possibility of operative interference, is very great. I have used this method for the past ten years.

About 1930 in the *American Journal of Cancer* (and I am sorry I can't give the man's name who devised the instrument or the exact title of the article) there was described a biopsy punch, a small trocar and cannula with a diathermy applicator which could be inserted through it to stop bleeding. I have used that frequently in tumors which approached the chest wall. By percussion one can safely locate the site of the tumor and put the small trocar in. This has a harpoon on the end of it which will surely draw out a piece of tissue large enough to be sectioned and examined.

DR. RALPH B. BETTMAN, CHICAGO, ILL.—I would like to sound a theoretical word of caution, and I am surprised that it has not been sounded before. We must realize that when we put a needle through an unopened pleural space and through lung into what may or may not be a tumor it is a dangerous procedure. There may be cases in which this procedure is justified (there undoubtedly are), but it seems to me we ought to go on record warning that there are very obvious dangers not only of spreading the malignancy, but of causing other damage in the course of the passage of the needle.

Certainly where the tumor is deep in the lung and where there are probably no adhesions between the pleura there cannot fail to be bad accidents with this method.

DR. J. SAMUEL BINKLEY, New York City.—I have not anything to add except that the Hoffman punch is probably the instrument that Dr. Head mentioned. We have not used the punch in recent years in this particular procedure. We believe that the 17 or 18 gauge aspirating needle has proved quite satisfactory.

In looking over the literature there were several case reports by Germans of fatalities in elderly arteriosclerotic patients due to perforation of intercostal arteries. This was before the time of the use of x-ray, and large trocars were used a great deal for the particular purpose of removing fluid. Occasionally a fairly large vessel may be injured by the use of the punch. We have been so satisfied with the ordinary aspirating needle that we prefer to use it, although the punch is a very good instrument in the hands of those who may prefer it.

A PORTABLE PNEUMOTHORAX APPARATUS*

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THE addition of this pneumothorax apparatus to the armamentarium of physicians has its justification in the portability, smallness of size, simplicity of construction, and ease of operation of this instrument.

The apparatus, complete for carrying, weighs only seven and one-half pounds. The case measures thirteen inches in height, eight inches in width, and five and three-fourths inches in depth.

CONSTRUCTION OF INSTRUMENT

The mechanics of this apparatus are quite simple. The gas that is delivered into the chest is filtered atmospheric air. The instrument consists of the following parts (Fig. 1):

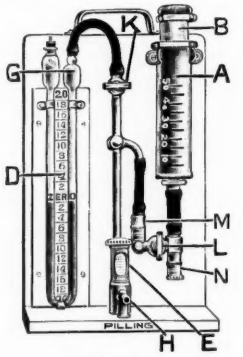

Fig. 1.

AB, syringe of 50 cu. cm. volume; *N,* a valve which permits atmospheric air to enter the syringe when the piston is drawn up, but will not permit air to escape when the piston is pressed down; *M,* a valve which closes when the piston is drawn up, thus preventing air from escaping from the pleural space into the syringe, and opens when the piston is pressed down permitting air from the syringe to enter the pleural space. *DG,* a water manometer for measuring the intrapleural pressures; *E,* a chamber in which air is filtered through gauze; *K, L,* two petcocks are introduced into the circuit to permit the operator to open or close various parts of the circuit as he desires.

*This apparatus is made by the George P. Pilling and Son Co., Philadelphia.

Received for publication Aug. 1, 1936.

†From the Eagleville Sanatorium, Eagleville, Pa.

METHOD OF OPERATION OF APPARATUS

In giving an insufflation the procedure is as follows: The needle is introduced into the chest in the manner usual to the operator. With the petcock *L* closed and *K* open the intrapleural pressure oscillations are determined. Then *K* is closed and *L* opened so that the manometer is closed off from the pleural space and the latter now communicates with the syringe. The piston of the syringe is then drawn to the 50 cu. cm. mark. The piston is then permitted to fall of its own weight, or slight pressure is made upon it, forcing 50 cu. cm. of air into the pleural cavity. This procedure is repeated until the desired volume of air has been introduced. At any time during the insufflation the intrapleural pressure may be determined by closing *L* and opening *K*.

DISCUSSION

The smaller amounts of gas that can be delivered by this apparatus by virtue of the small delivery chamber (50 cu. cm. volume accurately divided on the syringe) make it a useful instrument in giving the initial pneumothorax treatment, where close examination of the behavior of the intrapleural pressure with introduction of small quantities of air is desirable.

It may be felt that the weight of the piston forces air into the chest under too great pressure. However, the weight of the piston is largely overcome by the friction against the walls of the barrel. The volume of air introduced into the pleural space per minute, which is the important consideration in pneumothorax work, is no greater with this apparatus than with others, as I have determined by actual measurement.

CONCLUSION

A portable pneumothorax apparatus has been described.

NINTH AND RITNER STREETS
PHILADELPHIA, PA.

In Memoriam

John Lawrence Yates
1873—1938

Dr. John L. Yates died at his home at Milwaukee on November 3, 1938, after an illness of only three days which began with a bad throat infection.

He was president of the American Association for Thoracic Surgery in 1928 and since the first year of its organization he was a regular attendant at the meetings. In the earlier years of the Association's existence he was a frequent contributor to its programs.

He was one of a small select group of American surgeons in his day who were competent pathologists and of a still smaller group who were "physiologically minded." He overcame many obstacles to satisfy his urge to do original experimental work. Up to the time of his death he was still planning research problems. The scientific curiosity which he had was doubtless due not only to his special training in pathology, but also was the reason why he spent extra years in getting that training. He was born in Milwaukee, February 27, 1873, graduated from Yale in 1894 and from Johns Hopkins University Medical School in 1899. He became an assistant in pathology at Johns Hopkins in 1901 and was assistant demonstrator in pathology at the University of Pennsylvania under Dr. Simon Flexner until 1905. His special graduate training in surgery was received at Johns Hopkins under Dr. Halsted and later at Chicago for a year with Dr. Albert J. Ochsner. He began a surgical practice in Milwaukee in 1906. At the time of his death he was on the staff of the Columbia Hospital, of the Milwaukee Children's Hospital and of the County Hospital. He was also an associate research professor on tumors at the University of Wisconsin.

Probably his most important contribution was his careful experimental work which demonstrated for the first time the impossibility of satisfactorily draining the peritoneal cavity in cases of peritonitis, together with certain other suggestions about the treatment of that condition. This work at first was received with some skepticism by surgeons in general, but for it he was awarded the Senn Medal in 1905 and the Gross Prize in 1915. Only a few years were required to have his principal idea universally accepted. With Bunting, of the University of Wisconsin, he carried out an intensive study of Hodgkin's

disease and became the recognized authority on that subject. It was largely due to his demonstration of the futility of radical excision that the operative treatment of this condition was abandoned. During the war he held the rank of lieutenant colonel and saw much active service in France. In the latter part of 1918 he was actively engaged with the Red Cross group at Dijon, France, in experimental work on gunshot wounds of the chest, surgical shock, and other conditions. In 1923 he published an experimental study on the "Effects of Acute and Chronic Pneumothorax," in which he proposed the idea that distention of the capillaries of the lung enlarges the alveoli, and he felt that such a mechanism explains the increased resonance (Skoda's) adjacent to an area of pneumonia.

John Lawrence Yates

In addition to the AMERICAN ASSOCIATION FOR THORACIC SURGERY he was a member of the *American Surgical Association,* the *Clinical Surgical Society,* the *American College of Surgeons,* the *Chicago Surgical Society* and other medical societies.

Jack Yates had a delicious but kindly sense of humor which was a constant joy to his close friends. His amusing letters brightened many a dull day. By those who knew him well he was greatly loved, but by others he was often misunderstood. He was an idealist who was disappointed in his failure to realize his dreams. His standards of honor and integrity were of the highest. On several occasions in the

course of his experimental work he inoculated himself with the material which he was studying when he thought that a human experimental animal was needed. Once as a result of such an experiment upon himself several months were required for recuperation. As an operating surgeon he was careful and skillful, and his clinical judgment was of the best. He will be greatly missed by a large circle of friends both medical and nonmedical.

He is survived by his wife Katherine Gross Yates and a sister, Mrs. C. M. Allis of Milwaukee.

Evarts A. Graham.